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William J. Reals, M.D., Wichita, in private practice of pathology and Dean of the University of Kansas School of Medicine-Wichita, was elected to the American Medical Association Council of Medical Education at the AMA Interim Meeting in December.

Dr. Bill Reals has been a member of the Kansas Medical Society since 1950, and served as its president in 1971-72. An AMA member since 1949, he was Alternate Delegate from Kansas for nine years. He currently is the AMA Delegate from the College of American Pathologists.

Dr. Reals is the first Kansas physician to be elected to a major AMA position.

I don't want to run the cost up.

That's Good, Mel. We should all Opt to stop unnecessary health care costs.



Mel has just opted to take his doctor's advice that his surgery can be performed on an out-patient basis rather than his being hospitalized unnecessarily overnight.

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*Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic.

†All benzodiazepines, however, produce additive effects when given with CNS depressants, such as barbiturates or alcohol.

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Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS. It is not known if oral lorazepam is excrêted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® IV for (lorazepam) Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.


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Phenylephrine Hydrochloride	25 mg
Phenylpropanolamine Hydrochloride	50 mg
Chlorpheniramine Maleate	8 mg
Hyoscyamine Sulfate	0.19 mg
Atropine Sulfate	0.04 mg
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Ru-Tuss Tablets act continuously for 10 to 12 hours.

Ru-Tuss Tablets are an oral antihistaminic, nasal decongestant and anti-secretory preparation.

INDICATIONS AND USAGE Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues. Phenylephrine and phenylpropanolamine combine to exert a vasoconstrictive and decongestive action while chlorpheniramine maleate decreases the symptoms of watering eyes, post nasal drip and sneezing which may be associated with an allergic-like response. The belladonna alkaloids, hyoscyamine, atropine and scopolamine further augment the anti-secretory activity of Ru-Tuss Tablets.

CONTRAINDICATIONS Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.

WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

PRECAUTIONS Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with glaucoma, or urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See Warnings).

OVERDOSAGE Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS Hypersensitivity reactions such as rash, urticaria, leukopenia, agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerotomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.

DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.

HOW SUPPLIED:

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Expectorant

DESCRIPTION

Each fluid ounce of Ru-Tuss Expectorant contains:

Codeine Phosphate	65.8 mg
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Phenylephrine Hydrochloride	30 mg
Phenylpropanolamine Hydrochloride	20 mg
Pheniramine Maleate	20 mg
Pyrilamine Maleate	20 mg
Ammonium Chloride	200 mg
Alcohol	5%

Ru-Tuss Expectorant is an oral antifussive, antihistaminic, nasal decongestant and expectorant preparation.

INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief of upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms associated with hay fever, allergies, nasal congestion and cough due to the common cold.

CONTRAINDICATIONS Hypersensitivity to antihistamines. Concomitant use of an antihypertensive or antidepressant drug containing a monoamine oxidase inhibitor is contraindicated.

Ru-Tuss Expectorant is contraindicated in patients with glaucoma, bronchial asthma and in women who are pregnant.

WARNINGS Ru-Tuss Expectorant contains codeine phosphate, therefore, the patient should be warned of the potential that this drug may be habit forming. Ru-Tuss Expectorant may cause drowsiness. Patients should be warned of the possible additive effect caused by taking antihistamines with alcohol, hypnotics, sedatives and tranquilizers.

PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicle or operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease.

Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.

ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions.

DOSAGE AND ADMINISTRATION Adults: 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.

Children 6 to 12 years of age: ½ the adult dose, not to exceed 6 teaspoonfuls in any 24-hour period. Children 2 to 6 years of age: ½ teaspoonful every 4 hours, not no exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age: Use as directed by a physician.

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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Our thanks to Dr. Waxman and Dr. Reals for explaining in the last two issues how AMAERF funds are utilized by their schools. I hope you take this to heart and contribute generously to this important cause.

The past four months I have been on the road from Caney to points in all of Kansas — Colby, Protection, Garden City, Kansas City, Parsons, and others. I have visited 17 of the 27 auxiliary chapters and have enjoyed every one of them.

I took the auxiliary message via the slide presentation, "It Pays to Belong," to ten county chapters. This is a presentation of 80 slides demonstrating the various activities and projects of the American Medical Association Auxiliary. The narration that accompanies it explains that "it's an investment in the future with opportunities for involvement as varied as your interest; from planning and implementing community programs to working on legislative activities to raising funds for medical students and schools to developing your own management, organizational, and creative skills." If any of you physicians are in doubt as to what your auxiliary is and does, I will be glad to come and show the slides — just let me know!

Meldon Laury (David) was with me on most of the jaunts, but she was alone when she had the misfortune of hitting a deer as she was traveling Highway 50 east of Peabody. Fortunately she was not hurt, but Dr. Laury's car was without headlights and was out of commission for a few weeks.

Also with me on the tour of Kansas was the beautiful afghan crocheted by Cathy Mowry (Jerry), Manhattan, in wedgewood green and white blocks with various colored wild flowers embroidered in the blocks. She was generous indeed in donating it for our AMAERF project; we are taking dollars for names, one of which will be drawn from a hat at the convention in Wichita the night of the auction — April 30. For more information, see your local AMAERF chairman.

This month report forms will be distributed to the local presidents so we may evaluate all projects of the auxiliary chapters in Kansas. Also, we are drawing up plans for the state convention in Wichita and will divulge them next month.

Until then. . . .

Betty L. Moore,
President
Kansas Medical Society Auxiliary

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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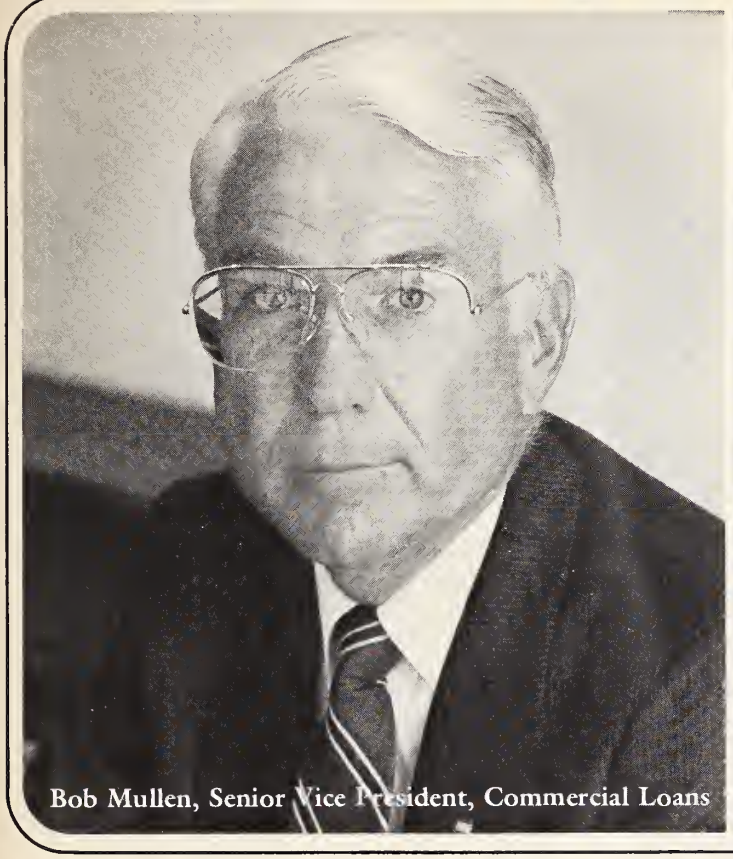
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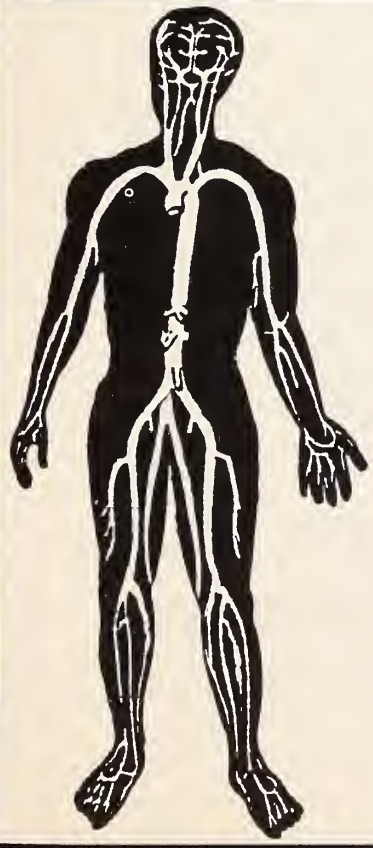
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RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
Robert P. Hudson, M.D., Kansas City ... (913) 588-7040
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
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Case Reports

STAPHYLOCOCCUS AUREUS BACTERIURIA ASSOCIATED WITH BACTEREMIA

Urinary tract infections are commonplace; however, Staphylococcus aureus is infrequently implicated as the pathogen.[1] In addition, the urinary tract is rarely the source of staphyloccal bacteremia.[2] Herein we report a case of Staphylococcus aureus bacteriuria associated with bacteremia.

Case Report

A white male 84 years old presented to Truman Medical Center with a four-day history of lower abdominal pain. Past history included a left hip fracture from which he was recovering. Because of urinary incontinence, a Foley catheter had been in place until four days prior to admission. After its removal he had been unable to urinate and his abdomen became progressively distended. Physical examination was unremarkable except for a respiratory rate of 45/min and a symmetrically distended lower abdomen. A Foley catheter was passed and approximately 650 ml of purulent urine was obtained. Pertinent laboratory values included a blood urea nitrogen, 70 mg/dl (normal 8-20 mg/dl); serum creatinine, 3.5 mg/dl (normal 0.5-1.0 mg/dl); and white blood cell count, 20,000 mm^3 with differential of 67 polys, 30 bands, and 3 lymphocytes. Arterial blood gases revealed hypoxemia and respiratory alkalosis. Urinalysis showed proteinuria, 4+; blood, 2+, white blood cells too numerous to count; and bacteria. Urine culture demonstrated Staphylococcus aureus, and four blood cultures from separate sites revealed the same organism. A diagnosis of urinary tract infection with bacteremia was made and appropriate treatment begun.

Discussion

Previous investigations indicate that Staphylococcus aureus is an uncommon uropathogen.[1] In a retrospective study, Demuth and colleagues found this organism in approximately 1 per cent of positive urine cultures.[3] However, there was a 5.5 per cent incidence of secondary bacteremia in their group of patients studied in a veterans hospital. Prior to their report there had been few previous cases associating Staphylococcus aureus bacteremia with a urinary tract source. In fact, the 5.5 per cent incidence of bacteremia in their study exceeds the occurrence of 2.3 per cent of secondary bacteremias from all nosocomial urinary tract infections demonstrated by the National Nosocomial Infection Study.[4]

Our patient demonstrates the clinical characteristics consistent with Staphylococcus aureus bacteriuria. Most patients are elderly, asymptomatic males who are afebrile and have pyuria on urinalysis. In addition, urinary tract manipulations or abnormalities are quite frequent. In Demuth's study, 73 per cent of the nosocomial infections were associated with urinary catheterization or other invasive urinary tract procedures.[3] However, in contrast to Demuth's group, in which 61 per cent of cases were not treated, our patient was treated because of the clinical suspicion of bacteremia. Staphylococcus aureus bacteriuria is not limited to elderly males, as shown in Demuth's group in which several patients were female and under age 20 years.

In summary, Staphylococcus aureus bacteriuria should be (1) a clinical clue that a urinary tract abnormality may be present; and (2) should be treated with specific antistaphylococcal therapy because of the risk of bacteremia. In addition, we suggest that prior to any diagnostic or therapeutic procedures, the urine be recultured in order to validate the diagnosis and avoid the possibility of contamination.

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EPIGLOTTITIS: LARYNGOSPASM AT INTUBATION

Mortality from acute epiglottitis caused by Hemophilus influenzae type B has fallen from 40 per cent in the preantibiotic era to 2 per cent in series reported today.[1] Management continues to evolve. Early elective nasotracheal intubation has become the preferrred method of establishing a reliable patent airway at many centers[2, 3] since extubation is possible within 48-72 hours and complications of tracheostomy are avoided.

In the case presented here, intubation of the patient was impossible due to the development of laryngospasm, necessitating an emergency tracheostomy. Possible explanations for this phenomenon are offered and a management plan suggested to minimize the possibility of laryngospasm.

Case Report

A 5-year-old white male was well until the morning of admission when he developed a fever and sore throat. Difficulty in breathing developed during the next few hours, and he arrived at the emergency room approximately eight hours after the onset of symptoms. Examination revealed a frightened, alert, cooperative boy sitting up with jaw thrust forward with muffled inspiratory stridor and mild intercostal retractions without cyanosis. Temperature was 39.4C; pulse, 130/min; blood pressure, 110/70 mmHg; respirations, 35/min; and weight, 16.2 kg. No attempt was made to visualize the throat or epiglottis. Results of the remainder of the physical examination were within normal limits.

Total white blood count was 30,000 with 40 neutrophils, 20 bands, and the remainder lymphocytes and monocytes. Lateral neck x-rays demonstrated ballooning of the hypopharynx, marked thickening of the aryepiglottic folds, and an enlarged poorly defined epiglottis.

Five per cent dextrose solution was administered intravenously, and the patient was taken directly to the operating room where he received 0.2 mg atropine, followed by 25 mg ketamine intravenously. At laryngoscopy, an enlarged red epiglottis was confirmed, and material was obtained for culture. Endotracheal intubation was attempted, but was impossible due to laryngospasm. Emergency tracheostomy was then performed.

Following the procedure, the patient was transferred to the intensive care unit. Intravenous chloramphenicol (100 mg/kg/24 hrs) and ampicillin (200 mg/kg/24 hrs) were begun following a blood culture which subsequently grew Hemophilus influenzae

type B sensitive to ampicillin. Following the sensitivity report, chloramphenicol was discontinued after 72 hours. Epiglottic cultures grew alpha Streptococcus and Enterobacter species. The tracheostomy tube was removed after 72 hours, and intravenous ampicillin was discontinued after four days. Oral ampicillin (100 mg/kg/24 hrs) was continued for another six days. Recovery was uneventful with hospital discharge occurring on day six.

Discussion

Acute epiglottis is not a common disease, accounting for approximately one in 3,000 pediatric admissions. It results from invasion of the epiglottis and other supraglottic tracheal tissues primarily, if not solely, by Hemophilus influenzae type B, resulting in toxicity, severe throat pain, and rapid development of airway and esophageal obstruction. Sudden unexplained complete airway obstruction can occur due to epiglottic swelling, obstructive secretions, and laryngospasm. Improvement in survival has developed as a result of effective antibiotics and discarding the "watch and wait" approach in favor of elective establishment of an airway immediately upon diagnosis in all cases with any degree of obstructive symptomatology.[1-3]

Intubation of a swollen trachea occluded by an enlarged epiglottis can be extremely difficult, but at Buffalo Children's Hospital, 45 of 48 children (94%) with epiglottitis and blood cultures positive for Hemophilus influenzae type B were successfully intubated. There were no unsuccessful intubations and no patient in the group required tracheostomy.[1]

Why then did our patient develop such an extreme degree of laryngospasm as to prohibit intubation? Manipulation of an inflamed epiglottis has produced abrupt laryngeal obstruction and death.[3] The laryngoscope blade probably touched the patient's epiglottis, and swabbing of the epiglottis for culture was done at laryngoscopy prior to intubation. However, there is another factor that may have contributed to the laryngospasm, a factor that has not been adequately addressed in the pediatric literature — the choice of anesthetic.

Our patient received ketamine, an anesthetic agent commonly used in children. Its advantages include the absence of respiratory depression if used in recommended doses and a wide margin of safety. However, ketamine has been associated with laryngospasm, and for this reason may well be contraindicated in epiglottitis. The following is from the Ketalar (ketamine) package insert provided by Parke-Davis dated July 1975. "PRECAUTIONS — Because pharyngeal and laryngeal reflexes are usually active, Ketalar should not be used alone in surgery or diagnostic procedures of the pharynx, larynx or broncheal tree. Mechanical stimulation of the pharynx should be avoided, whenever possible, if Ketalar is used alone. Muscle relaxants, with proper attention to respirations, may be required in both of these instances . . . Laryngospasms and other forms of airway obstruction have occurred during Ketalar anesthesia."

An alternative preferable to ketamine would be sedation with intravenous Valium with intubation while the child is near sleep, but still awake. Five patients were successfully intubated using a combination of thiopental, atropine, and succinylcholine.[2]

Given the current understanding of epiglottitis, the following plan of management is proposed:

- If a child presents with classic symptoms of epiglottitis, s/he should be taken immediately to the operating room with a minimum of disturbance (no blood drawing, no inspection of the throat, no x-rays, initiation of intravenous solutions in the operating room) for inspection of the epiglottis to confirm the diagnosis and for immediate nasotracheal intubation if a large red epiglottis is visualized.
- If the diagnosis is in doubt at presentation, soft tissue lateral neck films should be obtained with the physician in attendance, prepared to support ventilation if necessary. If the films support the diagnosis of epiglottitis, proceed to the operating room. If the films are normal, tongue blade inspection of the throat and epiglottis should be undertaken to avoid missing an early case.
- It is reasonable to observe a patient without intubation in the intensive care unit in the rare event that an enlarged red epiglottis is visualized as part of the evaluation of a sore throat, if there are absolutely no signs of airway obstruction, and if intubation is planned for the development of obstructive symptomatology.[1]
- Avoid ketamine. Select a sedative or anesthetic agent suitable for procedures requiring stimulation of the pharynx. Avoid touching the epiglottis with the laryngoscope blade and wait until an airway has been established before obtaining culture material from the epiglottis.
- Prior to induction of anesthesia, the operating room should be stocked with appropriate sized tracheostomy tubes for use in the event intubation is not possible. If a laryngeal foreign body is a possibility, appropriate sized bronchoscopes should also be on hand.
- Obtain a blood culture and perform other desired blood work after establishment of the airway. The risks of agitating a child to obtain a blood culture or to begin intravenous antibiotics prior to establishing an airway outweigh the benefits since total airway occlusion could result,[1] and the likelihood of metastatic Hemophilus influenzae type B disease is small despite the high rate (70-80%) postive blood cultures.[2, 4]
- Following blood cultures, initiate parenteral chloramphenicol and ampicillin to cover Hemophilus influenzae type B which might be resistant to either of these drugs,[5-7] with plans to discontinue the chloramphenicol if reports indicate sensitivity to ampicillin.
- Attentive care of the airway in the intensive care unit is mandatory until extubation is accomplished after 48-72 hours. An additional 24 hours of observation is indicated to be sure that signs of post-extubation subglottic edema do not develop. Antibiotics should be continued intravenously for at least 48-72 hours, then continued orally when the child is able to swallow, for a total course of ten days.[1, 3, 8]
- Prophylactic treatment of siblings[9] or contacts in closed populations such as day care centers[10] with rifampin[11, 12] should be considered.

Summary

A patient is presented who suffered acute epiglottitis due to Hemophilus influenzae type B in whom intubation was rendered impossible. Ketamine anesthesia may have contributed to the laryngospasm and is contraindicated in epiglottitis. A plan of management is suggested.

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PAINFUL TRAUMATIC NEUROMA OF THE VAGINAL CUFF

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Traumatic neuromas are frequently overlooked as a source of pain and are usually thought of as occurring in peripheral nerves. Much has been written about traumatic neuromas of the amputation stump, head and neck neuromas, intra-oral neuromas, cystic duct remnant neuromas, vagus nerve neuromas, intercostal nerve neuromas, interdigital neuromas, and neuromas of other nerves. In reviewing the literature, we find no report of painful

traumatic neuroma of the vaginal cuff; hence, this report deals with an unusual case.

Case Report

A 31-year-old white female complained of painful intercourse, pelvic pain, and some backache. This condition had existed for approximately five years, but had been more constant during the last two years. The patient's past history revealed that she had had an abdominal hysterectomy, bilateral salpingo-oophorectomy, and appendectomy at age 21 years. The operation was done for endometriosis and pelvic infection, and she tolerated it well.

Pelvic examination revealed good support of the vagina; uterus and ovaries were absent. At the top of the vagina on the left side was a small one-quarter inch slightly reddened tumor believed to represent granulation tissue. That region was quite tender. There was also some prolapse of the upper part of the vagina.

At surgery numerous adhesions were divided. The heavy scar tissue at the top of the vagina was removed along with the small tumor. A v-shaped portion of the upper vagina was removed, and the sacro-uterine ligaments were shortened. The vaginal cuff was closed transversely with catgut suture. She did quite well, and convalescence was uneventful.

The patient subsequently reported relief from pelvic pain and painful intercourse. Recent pelvic examination revealed no tenderness in the upper vagina.

The pathology report revealed normal appearing stratified squamous epithelium along the surface. Within the deep stroma were collections of tangled nerve fibers and bundles within a dense fibrous matrix *(Figure 1)*. A Bodian stain was positive for nerve fibers within the tangled bundles. A diagnosis of submucosal traumatic neuroma, probably secondary to previous surgical procedure, was made.

Mathews and Osterholm[1] have pointed out that neuromas are truly ubiquitous. They state that in every instance where a nerve is sectioned and cannot reestablish continuity with its distal counterpart, a neuroma of sorts results. Certainly many do not become painful. Hankin and Reed,[2] in defining a neuroma, state that it is a proliferative, non-neoplastic mass formed at the site of traumatic transection or crush injury of a nerve, composed of an entanglement of axons, Schwann cells, and fibrocytes in a dense collagenous matrix. They discuss neuromas in relation to peripheral nerves.

Hajdu,[3] in reviewing the case reported here, stated that the lesion did not qualify as a true neoplasm. He reports in his book[4] that neuromas are not true neoplasms but result from trauma-initiated proliferation of the axon-Schwann cell unit.



Figure 1

Summary

A case of painful traumatic neuroma of the vaginal cuff is reported. The literature has been reviewed; a neuroma in this location is extremely rare. This should be kept in mind in making a diagnosis in women with pain in the vaginal cuff area.

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LEGIONNAIRE'S DISEASE PRESENTING AS FULMINATING RESPIRATORY FAILURE

Presented at the Regional Meeting of the American College of Physicians in Kansas City, February 1980. This is the first reported case of confirmed antemortem diagnosis of Legionnaire's disease in the state of Kansas.

Legionnaire's disease is a potentially fatal illness. The disease is being increasingly recognized throughout the world as a cause of significant morbidity and mortality. This disease entity has protean clinical manifestations varying from mild self-limited febrile illness to a severe fulminant pneumonia with respiratory failure and multisystem involvement.

Reported here is the successful treatment of a patient in whom adult respiratory distress syndrome (ARDS) developed secondary to Legionnaire's disease. It was associated with mental confusion, obtundation, hepatic dysfunction, syndrome of inappropriate antidiuretic hormone secretion, hypophosphatemia, and evidence of microscopic hematuria. The purpose of this report is to stress the characteristic clinical pattern of the disease which may be distinctive enough to allow early diagnosis and institution of specific and perhaps life-saving therapy.

Case Report

A 47-year-old Caucasian male veteran was admitted to the intensive care unit with high fever and in acute respiratory distress. His health had been normal until three days earlier when he developed myalgia, malaise, and anorexia. The following day his temperature was 40 C, and he had chills with dry hacking cough. Chest x-ray showed a patchy, alveolar infiltrate in the left upper lobe, and he was started on ampicillin. The following day he experienced nausea, vomiting and watery diarrhea, and became profoundly weak. Spiking fever and shaking chills continued, and he became gradually confused and dyspneic. On the day of admission his temperature was 40.5C, and he had developed respiratory distress and was obtunded. Chest x-ray showed a greater degree of pneumonic infiltrate than two days previously *(Figure 1)*.

The patient was a resident in the Nursing Home Care Unit of the Topeka VA Medical Center for rehabilitation following a car accident two years earlier, but this condition had never caused any significant problems. He had smoked one pack of cigarettes per day for the past 30 years. He had no past history of significant cardiorespiratory disease and was not receiving any regular medications.

On admission the patient was acutely ill, toxic, diaphoretic, and in respiratory distress. He was disoriented and uncooperative but had no localizing neurologic signs. Temperature was 40.5C; pulse, 96/min; respirations 32/min; and blood pressure, 95/60 mmHg. The neck was supple. He showed signs of con-



Figure 1. X-ray at admission.

solidation over the left upper lobe, and a few rales in the right base with scattered inspiratory wheezes. There were no murmurs or gallops. Liver and spleen were not palpable, and no lymphadenopathy or cutaneous rash was noted.

Laboratory data showed hematocrit, 42% with normal indices, and a white blood count of 10,800 cubic mm with 55% polymorphonuclear leukocytes and 20% band forms. Prothrombin time and partial thromboplastin time were slightly prolonged, but the platelet count was normal. Sedimentation rate was 54 mm/hr. Total bilirubin was normal; liver enzymes were elevated; SGOT, 64 units/ml; SGPT, 96 units/ml; and LDH, 327 units/ml. Urinalysis revealed ten red blood cells, an occasional white cell and granular cast, but no red cell cast. The level of creatinine phosphokinase was 2400 I.U. without myoglobinuria. The levels of blood urea nitrogen, creatinine, calcium, and magnesium were all normal. Electrolytes were normal apart from serum sodium which was markedly low (112 mEq/L) with low serum osmolality (234 mOsm/L) and the urinary osmolality was 680 mOsm/L. Urinary sodium was 27 mEq/L suggesting inappropriate ADH. Serum phosphorus (inorganic) was low at 1.7 mgm/100 ml. Sputum gram stain showed a few clusters of polymorphonuclear leukocytes without bacteria being demonstrated and further cultures showed normal flora. Acid fast stains, as well as multiple blood and urine cultures, were repeatedly negative.

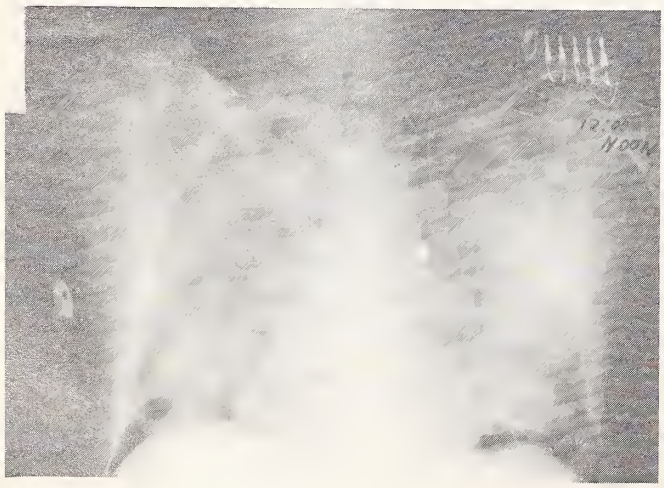


Figure 2. Third hospital day.

Initial arterial blood gas studies on supplemental oxygen 4L/min by nasal cannula showed pH, 7.54; carbon dioxide pressure (pCO_2), 23 mmHg; and oxygen pressure (pO_2), 52 mmHg. The patient was started on amikacin and cefoxitin intravenously, and ampicillin was discontinued.

During the following two days he remained pyrexial with recurrent shaking chills and dyspnea. Sputum production remained scanty but blood tinged. Chest x-ray showed an increase in infiltrate in the left upper lobe and a new infiltrate in the right lower lobe. Skin tests for tuberculosis and fungi were negative.

Extensive diagnostic workup for underlying etiology was performed; this included serial serological studies for mycoplasma pneumoniae, Legionnaire's disease, and respiratory viruses; also Q fever, tularemia and psittacosis. The cold agglutinins were negative as were all cultures of sputum, urine, and blood.

On the third hospital day, the patient's condition deteriorated rapidly. X-ray films of the chest showed that pneumonia had become pan-lobar *(Figure 2)* with extensive bilateral alveolar infiltrate with air bronchogram. Although the patient was receiving 100 per cent oxygen, his arterial oxygen pressure (pO_2) dropped to 44 mmHg. He was intubated and started on mechanical ventilation using PEEP, 10 mg H_2O and FIO_2 50%. Other supportive measures included continued fluid restriction, diuretics, and adequate monitoring of cardiac and hemodynamic status. In view of the high index of suspicion of Legionnaire's disease in that clinical setting, the patient was started on erythromycin intravenously, 4 gm/day.

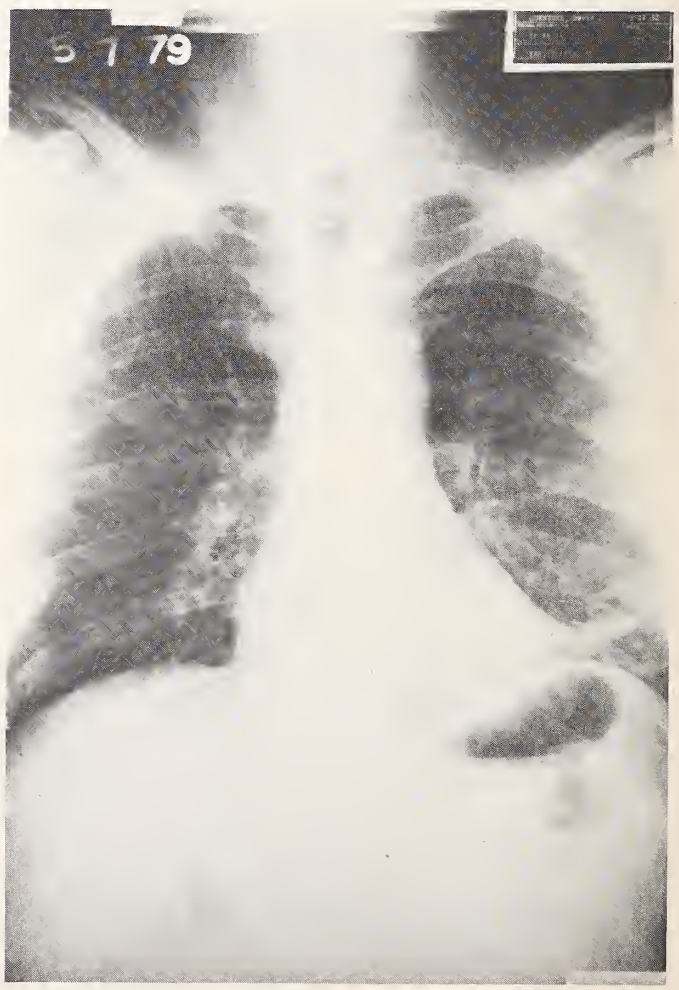


Figure 3. Fifteenth hospital day.

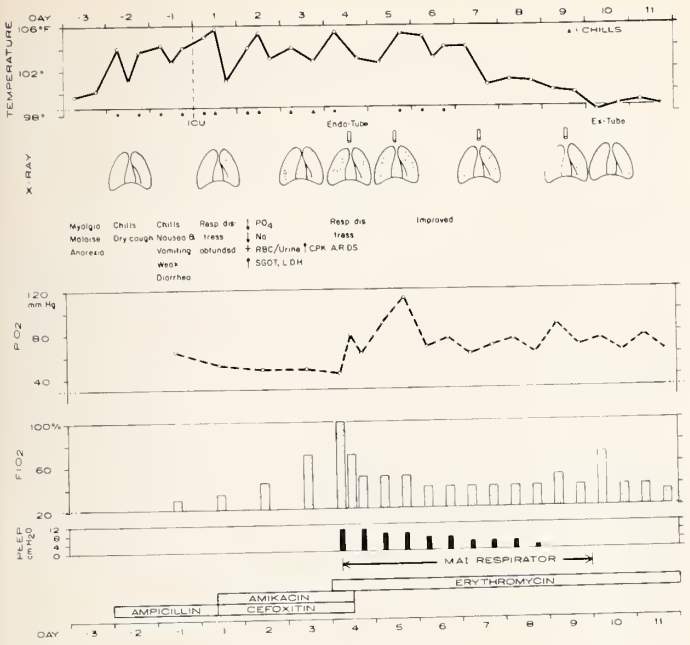


Figure 4. Progression of disease.

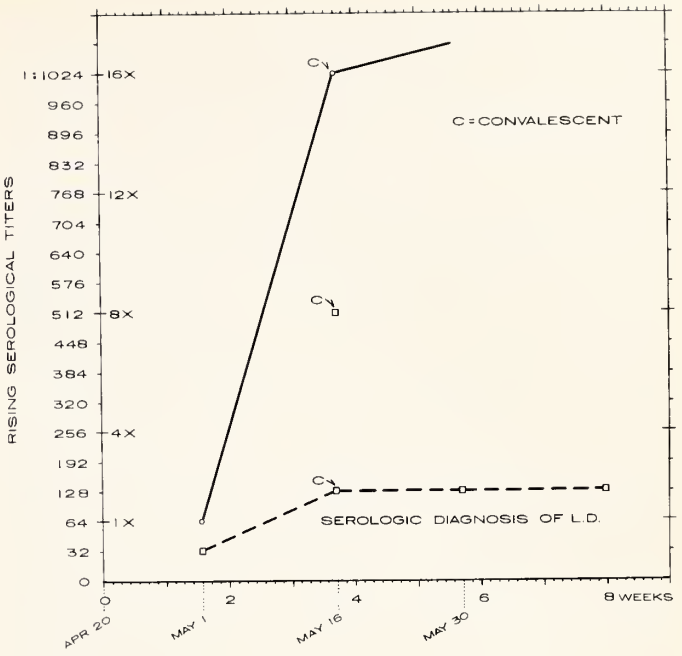


Figure 5. Serologic Diagnosis

During the next three days, the patient's respiratory status showed gradual improvement, and PEEP was reduced to 4 cm H_2O and FIO_2 40%. Blood gas studies showed pH, 7.44; pCO_2, 30 mmHg; and pO_2, 60 mmHg. The fever, which was unremittent, dropped by the end of the fifth day of erythromycin therapy. The patient was extubated after six days of mechanical ventilatory support. There was gradual resolution of pneumonic infiltrate on chest x-ray followup, and nearly complete clearing was noted on day 15 *(Figure 3)*. Therapy was erythromycin, 2 gm/day orally, and was continued for three weeks. The patient fully recovered. The overall progression of the disease is depicted in *Figure 4*.

Sera sent to the Center for Disease Control (Atlanta) for indirect fluorescent antibody testing demonstrated Legionnaire's disease (Serogroup 3) titers of 1 : 64 for serum drawn on May 1 (acute phase), with subsequent rises to 1 : 1024 on May 16 (convalescent phase), and greater than or equal to 1024 on May 20, indicating recent infection with Legionnaire's disease *(Figure 5)*. Other serologic studies for mycoplasma, Q fever, psittacosis, cytomegalovirus, influenza, and adenovirus were reported as negative. Pulmonary function tests, performed two and six months after the illness, showed considerable recovery with slightly abnormal spirometry and mild restrictive disease with low diffusing capacity. The patient's pO_2 on room air was 74.6, and showed considerable improvement (91 mmHg) after exercise, thus denoting only minimal pulmonary damage. Serial pulmonary function test results are given in *Table I*.

Discussion

Since the isolation of Legionnaire's disease bacterium in 1977,[1] and the development of serologic techniques[2, 3] to detect infection with the organisms, Legionnaire's disease has been implicated retrospectively and prospectively in epidemics,[4] case clusters,[5] and sporadic cases[6] occurring in the United States and abroad. Legionnaire's disease has a broad range of clinical manifestations from a mild febrile nonpneumonic illness[7] to a fulminant multilobular pneumonia[4] with adult respiratory distress syndrome. However, the newly emerging clinical spectrum is that of multisystem involvement. In two recent clinically conducted studies by Friedman *et al.*[6] and Kirby and colleagues,[8] multisystem manifestations of Legionnaire's disease involving the central nervous, hepatic, renal and gastrointestinal systems, and rhabdomyolysis were demonstrated. In fact, the presence of these extrapulmonary manifestations may suggest the diagnosis of Legionnaire's disease.

In this case, the disease was manifested by a fulminant multisystem illness involving extensive pneumonia with ARDS, confusion and obtundation, hepatic dysfunction, inappropriate ADH syndrome with hyponatremia, hypophosphatemia, and microscopic hematuria.

Difficulties in diagnosis of a sporadic case of Legionnaire's disease stems from the fact that many of its features are seen in other diseases[9, 10] such as pneumococcosis, mycoplasma,[11] psittacosis, Q fever and tularemia, as well as plague, influenza and other viruses, and in immunosuppressed patients[12, 13] with opportunistic infections such as Pneumocystis carinii.

TABLE I
PULMONARY FUNCTION TEST RESULTS
TWO AND SIX MONTHS AFTER RECOVERY
FROM LEGIONNAIRE'S DISEASE

		Predicted	*2 Mos*	*6 Mos*
Forced Vital Capacity (Liters)	FVC	4.29	2.58	2.90
Forced Expiratory Volume in One Second (Liters)	FEV_1	3.39	2.40	2.45
	Ratio	>70%	84%	85%
Max Mid Expiratory Flow (Liters per second)	MMF	3.33	2.65	2.30
Total Lung Capacity (Liters)	TLC	5.99	3.85	3.80
Residual Volume (Liters)	RV	1.70	1.00	0.89
Diffusing Capacity (ml/min/mmHg)	DL_{CO}	26.66	15.71	16.90
Arterial Oxygen Tension (mmHg)	pO_2	>80	71.6	74.6
Arterial Carbon Dioxide Tension (mmHg)	pCO_2	<44	38.0	42.0

Because of the time required for sero conversion and the difficulty in isolation [14] or demonstration of the organism,[15] clinical recognition is imperative for the diagnosis of the acute disease. No single aspect of the disease is unique, but the clinical and laboratory features of Legionnaire's disease taken in the aggregate may be sufficiently characteristic to allow an early tentative diagnosis. Such characteristic features include the presence of high fever, with recurrent rigors and relative bradycardia, and the presence of initial diarrhea.[16] Rapid radiologic progression of a patchy pneumonitis to a lobar or multilobar consolidation, in spite of conventional antibiotic therapy, would be highly suggestive of the disease.[17] The presence of a fulminant bacterial pneumonia in the absence of significant sputum production and negative microbiologic studies makes the diagnosis of Legionnaire's disease very likely (especially on a trans-tracheal aspirate[8]). The occurrence of neurologic abnormalities[18] when associated with pneumonia should always suggest the possibility of Legionnaire's disease. These include somnolence, delirium, ataxia, obtundation, confusion, amnesia, visual hallucinations, and seizures.

The presence of hyponatremia,[19] due to syndrome of inappropriate antidiuretic hormone secretion, occurs in Legionnaire's disease as in other types of bacterial pneumonias. Hypophosphatemia[8, 20] not due to any of the usual causes of this abnormality was noted in recently reported cases of Legionnaire's disease. Its significance is unknown but it has been reported to occur in the course of other gram negative rod bacteremias.

Laboratory evidence of liver function abnormality was mentioned to be common in Legionnaire's disease. It is possibly related to the acute illness and its impact on the liver.[21] Normal function returns slowly with therapy or resolution of the disease.

The occurrence of myoglobinuria[22] in association with severe myalgias has been mentioned in Legionnaire's disease and may be due to acute rhabdomyolysis. Microscopic hematuria[4, 8, 23] of minor degree has been reported in up to 10 per cent of patients with Legionnaire's disease. Renal failure due to tubulointerstitial nephritis was reported in a minority of patients.

Severe cases of Legionnaire's disease have been complicated by disseminated intravascular coagulation (DIC)[24] with thrombocytopenia, elevated fibrin split products, and prolonged prothrombin and partial thromboplastin time. However, subclinical forms of DIC are relatively more frequent.

Many patients with Legionnaire's disease develop respiratory distress concomitant to the progression of pneumonia and show evidence of hypoxemia, hypocapnia, and respiratory alkalosis. These usually respond to oxygen therapy by conventional methods. However, some Legionnaire's disease patients develop features of ARDS with progressive life threatening hypoxia. A study of the Philadelphia epidemic showed that respiratory distress and hypoxia necessitated oxygen therapy for 62 of 119 patients and mechanical ventilatory assistance for 19. Only six patients recovered from respiratory failure with ventilatory assistance and the mortality rate in that group was 68 per cent. However, in recent years, with better understanding of the pathophysiologic mechanism of ARDS,[25] better utilization of PEEP, fluid volume management and proper maintenance of perfusion through adequate hemodynamic monitoring, the prognosis in such critically ill patients has improved considerably.

Our patient, after an initial febrile illness of three days, had an alarmingly rapid progression of pneumonia. By the fourth hospital day, both lung fields were completely opacified and assisted mechanical ventilation was required. The difficulty with oxygenation was severe and indicated marked shunting as shown by the wide (A-a) gradient. PEEP was required for three days and assisted ventilation for six days.

In the majority of infections with Legionella diagnosis has been made by demonstrating sero conversion or sero positivity.[10] A four-fold or greater rise in serum indirect fluorescent antibody (IFA) titers to a level of 1 : 128 or greater is considered diagnostic of Legionnaire's disease. A single convalescent titer of 1 : 512 is diagnostic in a patient with compatible illness. Our patient was diagnosed by this method and showed a 16-fold rise in acute and convalescent titers.

Other modalities for definitive diagnosis are less widely used or not yet generally available.[27] Cultures of Legionella[14] have been notoriously difficult and the organism has been isolated only rarely from clinical specimens. Visualization of the organism in tissue by the use of special stains[15] (Dieterle Silver impregnation stain or Gimenez stain) although valuable, are time consuming. Demonstration of the bacterial agent by direct immunofluorescence (FA)[26] is the most promising modality for early, specific, and rapid diagnosis of Legionnaire's disease. Using this technique, Sarovaletez and coworkers[13] were able to demonstrate the Legionnaire's disease bacterium in sputum, pleural fluid, and bronchial washings. However, in spite of its value, it is not yet in common use and the diagnosis of Legionnaire's disease remains a retrospective one.[28]

Based on clinical and laboratory data, erythromycin is at present the drug of choice.[29, 30] In our patient, there was significant clinical improvement after 48 hours, and the patient was afebrile on day five of erythromycin therapy. Institution of treatment should be prompt on clinical grounds alone, since laboratory diagnosis is usually not possible for several weeks. Erythromycin should be continued for at least three weeks to prevent relapse. Rifampin, although considerably effective in vitro and vivo, should not be used in treatment of unconfirmed cases because of the propensity for resistance to develop among many baceterial species. It should be held in reserve for proven cases that are not responding satisfactorily to erythromycin or when pulmonary abscesses are present.[31]

Although survivors of Legionnaire's disease pneumonia have delayed but complete radiologic clearing, some patients may develop residual changes as shown by pulmonary function testing. In a recent study,[18] the diffusing capacity (as measured by the carbon monoxide single breath method) was significantly reduced in survivors of the Philadelphia epidemic two years later. Interestingly, our patient demonstrated this abnormality two and six months after the acute illness *(Table I)*.

In conclusion, we report a sporadic case of Legionnaire's disease which had a fulminant course with features of ARDS which responded to adequate management. Difficulties in diagnosis of sporadic cases arise from the fact that sero conversion occurs several weeks after onset of the illness. Therefore, the clinician must depend on a high index of suspision in the appropriate setting for prompt, specific, and perhaps lifesaving therapy.

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References are available from the authors.

INFLAMMATORY HEMOBILIA

Hemobilia or hemorrhage into the biliary tract is a relatively rare disorder that has seen an increase in incidence during the past several years because of a growing number of traffic accidents causing liver trauma. Inflammatory hemobilia is still a very rare occurrence, and although ascaris infestation is probably the

leading cause of inflammatory hemobilia worldwide, biliary tract stones are the leading cause of this disorder in the United States. The following case report deals with a case of inflammatory hemobilia and the devastating consequences that may ensue.

Case Report

A 55-year-old male was transferred to Wesley Medical Center after a one-week course of fever, right upper quadrant pain, and jaundice which had not subsided on ampicillin therapy. This patient was one year status post-cholecystectomy and common duct exploration for choledocholithiasis and ascending cholangitis. The patient's postoperative T-tube cholangiogram at that time was read as normal, and the patient did well for several months until the present complaints began.

On admission, physical examination revealed a well-developed jaundiced male who was noted to have bilateral basilar rales, right upper quadrant tenderness, and hepatomegaly. Total bilirubin was 7.9. The patient was begun on intravenous cefamandole and intensive respiratory toilette. Liver-spleen scan on admission revealed defects suggestive of multiple hepatic abscesses. Exploratory laparotomy and common duct exploration revealed multiple common duct stones with evidence of cholangitis. Cultures revealed pseudomonas infection, and the patient was subsequently begun on gentamycin and carbenicillin.

Forty-eight hours postoperatively, the patient developed severe edema, hypernatremia and upper gastrointestinal hemorrhaging, and coagulation was found to be abnormal. These abnormalities were felt to be due, at least in part, to the carbenicillin, which was stopped, and the patient was subsequently scheduled for endoscopic retrograde cholangiopancreatogram (ERCP) which revealed hemobilia. A blood clot in the proximal T-tube had prevented egress of blood from the T-tube. Because of continued upper gastrointestinal bleeding, repeat exploratory laparotomy was performed with incision and drainage of abscess cavities and suture ligation of individual bleeding points. Seventy-two hours after the second exploratory laparotomy, the patient developed a spontaneous duodenal perforation which was treated with a gastrojejunostomy. Forty-eight hours later patient developed hemoperitoneum secondary to spontaneous hemorrhage of the right liver lobe. This was once again treated with exploratory laparotomy and suture ligation of bleeding points. The patient suffered no more bouts of hemobilia. However, during the following days and weeks, the patient developed a progressively severe renal failure and liver failure. Despite renal dialysis and appropriate hyperalimentation, the patient's condition worsened and he died on the 62nd postoperative day.

Discussion

In 1948, Sandblom suggested the term hemobilia to designate hemorrhage into the bile duct as a result of trauma to the liver. However, the cause of hemobilia is not confined to trauma. Other causes include inflammation, gallstone disease, neoplasms, and vascular disease. Classically a patient with hemobilia will experience a triad of biliary colic, gastrointestinal hemorrhage, and jaundice approximately three to four weeks after injury, although the timing is quite variable ranging anywhere from four days to five months.

Inflammatory hemobilia has numerous causes, including chronic cholangitis and pericholangiolar abscesses secondary to parasites, amoebic abscesses, tuberculosis and gallstone disease, hemorrhagic cholecystitis, or stricture or carcinoma of the biliary tract. Inflammatory hemobilia appears to develop around periductal abscesses with purulent cholangitis.

As with other forms of hemobilia, the diagnostic test of choice is selective arteriography. Although the ERCP is an effective alternative, it does not provide the anatomical specificity of arteriography.

The treatment of hemobilia depends to a degree on the cause. The simplest treatment consists of unroofing the cavity with suture ligation of bleeding points and adequate drainage. Simply packing the cavity alone has a high incidence of recurrence and is mentioned only to be condemned. Should conservative measures fail, segmental or lobar resection of extensive lesions can be utilized. This is considered the more definitive procedure but is associated with higher morbidity and mortality rates. This procedure should be reserved for larger lesions or those not controlled by any other means. Arterial ligation is still a controversial area. On one hand, its use in the treatment of hemobilia has been, to quote Fish and Nippert, "surprisingly effective." On the other hand lies the concern that any arterial ligation complete enough to stop the hemobilia must also lead to hepatic ischemia with subsequent necrosis. However, it was noted by Berne that hepatic artery ligation is safe and effective if performed proximal to the gastroduodenal artery. In their opinion, hemobilia occurs only in the presence of normal intrahepatic pressure, and reduction of this pressure to the low level maintained by collateral flow through the gastroduodenal artery has proved effective in curbing further bleeding from the hepatic cavity. In their opinion, the risk of significant hepatic ischemia was patently low, and abnormal liver function tests postoperatively were transient and mild. Although caution is urged in resorting to arterial ligation, it is believed that this should precede major hepatic resection when dealing with traumatic hemobilia. However, as this case report demonstrates, inflammatory hemobilia of the liver does not readily lend itself to resection as a treatment modality because of the bilateral nature in which the disease oftentimes presents itself. However, it should be noted that arteriography is still highly recommended as a useful adjunct to localize the bleeding site. Another treatment mode not often discussed is non-operative management. No clear indications for non-operative management of hemobilia have been established. Some authors have used the SGPT and SGOT ratios to predict bleeding episodes. Lesions that lie on the distal branch of the hepatic artery are among those with the best chance to heal, but those situated on large proximal branches should be dealt with surgically. It is stated that one may consider temporizing in selected cases where bleeding is not massive, but patients must be followed with arteriography. The non-operative modality is also, as a rule, unsuitable for the treatment of inflammatory hemobilia because of the infectious nature of the disease and the need for drainage of abscessed cavities. Mortality rates for patients treated non-operatively for multiple macroscopic liver abscesses without hemobilia range from 85-100 per cent.

It is our belief that unless the inflammatory process is well localized, hepatic resection and conservative management — which may be curative procedures in selected cases of traumatic hemobilia — are not acceptable alternatives in the treatment of inflammtory hemobilia. Management in these cases should consist of abscess drainage, suture ligation of individual bleeding points, biliary decompression, and intensive therapy.

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A list of suggested reading is available from the authors.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Hypergastrinemia and Zollinger-Ellison Syndrome

CHARLES L. BROOKS, M.D. *and* MARK S. McPHEE, M.D.,*
Kansas City, Kansas

In 1905 Edkins discovered a substance derived from extracts of antral mucosa that stimulated gastric acid secretion. This substance was not proven to be a distinct circulating hormone until 1964, when Gregory isolated it in pure form, determined its polypeptide structure, and synthesized it. The hormone so characterized is now known as gastrin.

Biochemistry

All biologic activity of gastrin is located at the C-terminal portion of this polypeptide molecule. The potency of the hormone as a stimulator of acid secretion may be increased by altering or blocking the N-terminal portion, making the molecule more resistant to degradation. Naturally occurring gastrin exists in five separate forms. In addition to "big" gastrin (the heptadecapeptide), "little" gastrin and "minigastrin," two other forms of gastrin exist: "big-big" gastrin and "component I." The first three are the predominant circulating forms of gastrin. Little gastrin is the most potent form biologically, but comprises only 10-30 per cent of total serum gastrin. Big gastrin comprises the majority of total gastrin as measured by radioimmunoassay (RIA). Gastrin assays by RIA are about 1,000 times more sensitive in detecting the hormone than the bioassay originally used.

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Physiology

Gastrin is normally secreted by gastrin-cells (G-cells) in the gastric antrum and duodenum. The G-cell surface contains receptors for stimulation and inhibition of gastrin release. Mechanisms normally resulting in gastrin release include luminal peptide and amino acid stimulation, gastric distension, antral alkalinization, vagal cholinergic influences, and stimulation by circulating calcium and epinephrine. Gastrin release from G-cells is normally inhibited by luminal acid and circulating secretin, gastric inhibitory peptide (GIP), vasoactive intestinal peptide (VIP), glucagon, or calcitonin. Gastrin has many demonstrable effects in pharmacologic doses, but its only known physiologic actions include stimulation of gastric acid, pepsin, gastric intrinsic factor, and pancreatic enzyme secretion. In addition, the large amount of circulating gastrin sometimes found in gastrinoma patients can act as a trophic factor to produce hyperplasia of the gastric mucosa and can also inhibit gastric emptying. The hormone is metabolized primarily in the kidneys, the principle route of excretion in the absence of renal failure.

Hypergastrinemic States

The differential diagnosis of hypergastrinemia is conveniently divided into disorders with or without evidence of associated acid hypersecretion by gastric analysis *(Table I)*. Acid hypersecretion into the lumen of the stomach normally triggers a negative feedback mechanism which suppresses further gastrin release by antral G-cells. When acid hypersecre-

TABLE I
CAUSES OF HYPERGASTRINEMIA

With acid hypersecretion:
Zollinger-Ellison syndrome (ZES)
Antral G-cell hyperplasia (pseudo-ZES)
Duodenal ulcer (familial ulcer disease with hyperpepsinogenemia)
Short bowel syndrome (massive small bowel resection)
Isolated retained gasric antrum (post-surgically)
? Partial gastric outlet obstruction
Without acid hypersecretion:
Gastric ulcer
Gastric carcinoma
Pernicious anemia
Atrophic gastritis
Rheumatoid arthritis
Age
After vagotomy and pyloroplasty
With or without acid hypersecretion:
Chronic renal failure
Pheochromocytoma
Hyperthyroidism
Hyperparathyroidism

tion is present, it may derive from failure of this negative feedback mechanism, or from primary uncontrolled gastrin secretion by pancreas-derived gastrinoma cells (Zollinger-Ellison syndrome). There are disease states in which hypergastrinemia without acid hypersecretion is present, resulting in achlorhydria or hypochlorhydria. This abnormality is accompanied by increases in the basal serum gastrin level as a "physiologic" response to decreased or absent gastric acid secretion. In these conditions, the G-cells produce and release gastrin in a relatively uncontrolled but physiologically normal fashion, since negative feedback regulation by gastric acid bathing the antrum is absent. Chronic renal failure may elevate the serum gastrin level via two mechanisms: (1) an impaired negative feedback loop with chronic gastritis producing a blunting of the normal gastric acid responses; and (2) decreased renal excretion of the hormone. Pheochromocytoma, hyperthyroidism, and hyperparathyroidism also increase circulating gastrin levels, presumably via direct catecholamine or calcium stimulation of the G-cells.

The majority of hypergastrinemic states show only moderate elevations in fasting serum gastrin level, *i.e.* 200-500 pg/ml with a normal range in most commercial assays of approximately 50-180 pg/ml. Zollinger-Ellison syndrome (ZES), pernicious anemia, gastric carcinoma (especially carcinomas involving the body of the stomach), and chronic renal failure are the only disease states known that may elevate serum gastrin levels to more than 1,000 pg/ml. However, about half of ZES patients have gastrin levels in the "gray" (200-500 pg/ml) range rather than in the "clear-cut" (more than 1,000 pg/ml) category.

Clinical Features of ZES

ZES was first described in 1955 as a triad of fulminant peptic ulcer disease associated with marked gastric acid hypersecretion and a non-beta cell pancreatic tumor. Due to recent advances in the sensitivity of the gastrin assay and the introduction of provocative tests, the majority of ZES patients are now being diagnosed prior to the onset of severe, recurrent gastroduodenal ulcerations and resultant multiple surgical procedures which once characterized the disease.

Males comprise 60 per cent of ZES patients with a peak incidence in the sixth decade. The primary tumor is usually found in the pancreas but occasionally (10%) may be located in other intra-abdominal sites. Metastatic lesions are common, especially in regional lymph nodes and liver. Peptic ulceration is present in greater than 90 per cent of cases. Ulcers distal to the duodenal bulb are present in up to 25 per cent. Multiple ulcers are occasionally present, and irregular thick gastric mucosal folds may be observed. Diarrhea due to a massive volume of gastric acid secretion, and to acid inactivation of pancreatic enzymes and bile salts may be clinically significant in 30 per cent. Fewer than one-half of patients have an identifiable multiple endocrine neoplasia syndrome (*i.e.*, MEN-I with adenomas of pancreas, pituitary and/or parathyroid). Additional history often reveals weight loss or previous gastrointestinal surgery.

Diagnosis

The diagnosis of ZES is made by demonstrating: (1) an elevated fasting serum gastrin level (it may rarely be normal or near-normal); (2) gastric acid hypersecretion via gastric analysis and, if necessary; (3) a positive secretin stimulation test for serum gastrins. The first clue to the diagnosis of ZES is usually clinical evidence of acid hypersecretin; that clue should be further investigated by obtaining a fasting serum gastrin level (*Table II* provides a list of indications for obtaining a specimen for serum gastrin). Basal acid outputs of 15 mEq/hr or more are characteristic, as are basal acid output (BAO) to maximal acid output (MAO) ratios greater than 0.6. ZES is the only known disease state that will produce a positive secretin stimulation test for gastrin, and

TABLE II
INDICATIONS FOR OBTAINING A FASTING SERUM GASTRIN LEVEL

Recurrent or multiple peptic ulcers
Peptic ulceration distal to first portion of duodenum
Persistent duodenal ulcers unresponsive to medical therapy
Large quantity of antacids consumed without relief of dyspepsia or epigastric pain
Prior to contemplated surgery for peptic ulcer disease (PUD)
Recurrence of ulcers after PUD surgery
Family history of ZES
Family history of MEN-1
Pancreatic islet cell tumor
Diarrhea of unknown etiology
Marked gastric acid hypersecretion documented by gastric analysis
Diabetes mellitus with PUD
Hypercalcemia with PUD

TABLE III
SECRETIN STIMULATION TEST

Indications:
Moderately elevated fasting gastrin (300-1,000 pg/ml)
Strong suspicion of ZES with a normal or only somewhat elevated fasting gastrin (50-300 pg/ml)
Previous gastric surgery with suspicion of ZES
Family history or suspicion of multiple endocrine neoplasia
Documented massive acid hypersecretion by gastric analysis
Protocol:
Use GIH or KABI secretin since they are chemically pure. Boots secretin may itself contain enough gastrin to produce a false positive elevation in serum gastrin levels. Also if an allergic or atopic history is present, skin testing should be carried out prior to IV Boots secretin injection
Draw fasting gastrin then serial gastrin levels at 2, 5, 10, 15, 30, 45 and 60 minutes after an IV bolus of 2 u/kg secretin
Interpretation:
Positive test is interpreted as a rise of gastrin of >100 pg/ml from fasting level
Few false positives reported; false negatives <10% if pure secretin used

this test *(Table III)* may be useful in differentiating ZES from other hypergastrinemic states, especially antral-G cell hyperplasia. Other procedures potentially useful in making this distinction are the calcium infusion test and protein meal stimulation test.

ZES is characterized by a marked increase in serum gastrin levels following the intravenous administration of secretin, which apparently stimulates the pancreas-derived gastrinoma cells to release gastrin but has little effect on antral G-cells. On the other hand, administration of a protein meal may produce a marked increase in serum ganstrins in antral G-cells hyperplasia (pseudo-ZES), while the gastrinoma cells of true ZES are not much affected by meal stimulation. Intravenous infusion of 4-5 mg/kg/hr of calcium in an appropriate solution during a 3-4 hour period may produce an increase in serum gastrin levels in both disease states, but the magnitude of increase should be much greater (more than 400 pg/ml from fasting levels) in true ZES than in antral G-cell hypersecretion.

Other procedures which may be performed in an effort to localize a true gastrinoma include an upper gastrointestinal series, an abdominal CT scan, and upper gastrointestinal endoscopy. These procedures are of uncertain clinical value in ZES, but may help to identify potentially resectable tumors limited to the duodenal wall or the tail of the pancreas. Standard mesenteric angiography may be useful in some patients in an attempt to localize a gastrinoma by "tumor blush." A new technique of transhepatic portal vein catheterization also shows some promise in localizing gastrin-secreting tumors, but remains experimental. In this procedure, gastrin levels are measured in various draining veins of the portal system, and the results for each location are compared with each other and with a simultaneously drawn sample from the hepatic vein. A gradient between one or two measurements and the hepatic vein reference suggests that the tumor may be secreting gastrin from that particular anatomic segment.

Treatment

The treatment of choice for ZES prior to the "cimetidine era" was total gastrectomy, in which the target source of gastrin-stimulated acid hypersecretion is entirely removed. The operative mortality for this procedure is 5-10 per cent or more, and perioperative morbidity seriously infringing upon the patients' quality of life may occur in more than 50 per cent of patients subjected to total gastrectomy. In addition, surgical efforts to resect or debulk the gastrinoma usually have proved quite disappointing clinically. For these reasons a new approach to the treatment of ZES was sought and found when cimetidine became available. Cimetidine (Tagamet) administration provides a partial "medical gastrectomy" by blocking the histamine H_2 receptor on the gastric acid-secreting parietal cell, thus eliminating much of the gastrin-stimulated acid hypersecretion.

Medical Treatment. Once ZES has been diagnosed, appropriate patients should be begun on a program of cimetidine therapy which must continue for the remainder of the patients's life unless surgical intervention (resection of lesion, total gastrectomy, or vagotomy) occurs. The goals of cimetidine therapy are: (1) to reduce or eliminate symptoms and complications; and (2) to reduce basal acid secretion to less than 10 mEq/hr during the entire dosing interval. The dose and the dosing interval required to reach these goals will vary from patient to patient. In order to document adequate suppression of acid secretion, hourly gastric analysis is required for the entire dosing interval (usually six hours) following the administration of a given amount of cimetidine. The dose or dosing interval should be adjusted accordingly to assure an acid secretion rate of less than 10 mEq/hr for the entire dosing interval. Failure to suppress acid secretion below 10 mEq/hr during the first two hours usually indicates inadequate dose, while successful initial suppression which shows rebound above 10 mEq of acid per hour in the fourth or fifth hour often indicates a need to change the dosing interval to every four hours (six daily doses). Inadequate dosage or interval are common causes for medical treatment failure in ZES patients. The cimetidine dosage requirement may change with time, so periodic gastric analyses of patients on cimetidine should be performed to document adequate dosing.

Medical therapy of ZES may also include liquid antacids administered at doses of up to 30 ml one and three hours after meals and at bedtime, and every two hours between meals. Mylanta II, Maalox Therapeutic Concentrate, or another equivalent antacid with high neutralizing capacity should be used. In addition, oral anticholinergics may be therapeutically useful in combination with cimetidine, especially for ZES patients in whom cimetidine requirements, as documented by gastric analysis on cimetidine, exceed 2.4 grams daily. Painful gynecomastia and other adverse side effects of cimetidine often occur at or above this daily dose. A long-acting anticholinergic may allow reduction of the cimetidine dosage to acceptable levels. Again, gastric analysis during therapy should be used to determine the correct dosage of anticholinergic/cimetidine combination. Gastric acid suppression below 10 mEq/hr for the entire dosing interval is the goal. Anticholinergic drugs probably should not be used in ZES patients who have had surgical vagotomy, since severe impairment of gastric emptying may result.

None of the medical management options thus far discussed has any direct therapeutic effect on the primary process, *i.e.* the gastrinoma itself. Chemotherapeutic agents — such as streptozotocin — which have some activity against other pancreatic islet cell tumors have been investigated, but the results to date have not been encouraging. Attempted chemotherapeutic treatment of metastatic, diffuse, or multiple gastrinomas remains largely experimental.

Surgical Treatment. The majority of ZES patients enjoy rapid and prolonged improvement with appropriate medical therapy. On the other hand, surgical management in this disease has certainly not been rendered obsolete by the advent of cimetidine. The option of attempting primary excision of the gastrinoma for cure occasionally provides surgical intervention with a strong advantage over medical management in ZES. The possibility for such a surgical cure is highest if a nonmetastatic gastrinoma is found in the tail of the pancreas or duodenal wall. Unfortunately this subset represents only 10 per cent or so of ZES patients and usually requires intraoperative confirmation or exclusion, since preoperative diagnosis is difficult. Recent reports suggest that vagotomy (highly selective or truncal with pyloroplasty) in combination with cimetidine markedly decreases gastric acid secretion in ZES as a long-term effect. In addition, vagotomy may allow a decrease in the cimetidine dose necessary to control acid hypersecretion, and also provides the surgeon with an opportunity to explore the tail of the pancreas and duodenal wall in search of a lesion suitable for primary excision and potential cure. Vagotomy/antrectomy and other partial gastric resections remain inappropriate procedures in almost all ZES patients since the incidence of refractory stomal ulceration postoperatively is extremely high. Current indications for total gastric resection include any of the following, especially after vagotomy or other prior gastric surgery: (1) failure to suppress ZES complications or symptoms on adequate medical therapy; (2) patient refusal of life-long cimetidine therapy; (3) patient non-compliance with medical therapy; or (4) unacceptable drug side effects from the medical treatment regimen.

Summary

Gastrin is a polypeptide hormone normally excreted in various forms from the G-cell population of the gastric antrum. Stimulation of gastric acid secretion is its primary physiologic action. Appropriate elevations of serum gastrin occur in the presence of hypochlorhydria. Inappropriate hypergastrinemia may be caused by: (1) an impaired feedback mechanism; (2) a gastrin-secreting tumor; (3) catechola-

TABLE IV
THERAPEUTIC APPROACH TO ZES

Inform patient of *all* the alternatives!
Cimetidine 300 mg qid with antacids. 6-hr gastric analysis following cimetidine administration
Adjust dose and interval so that acid secretion <10 mEq/hr during the entire dosing interval
Consider anticholinergic treatment if acid secretion >10 mEq/hr when approaching 2.4 g/day of cimetidine
Possible exploratory laparotomy with vagotomy and excision of pancreatic tail or duodenal wall lesion if present
Total gastrectomy if poor compliance or failure while on *adequate* medical therapy

mine stimulation; or (4) decreased renal excretion. *Table II* lists the principal indications for obtaining a serum gastrin level in clinical practice. The treatment of ZES has changed drastically during the past five years,and a brief treatment protocol is provided in *Table IV*.

Self Assessment Questions

1. Besides ZES, name three hypergastrinemic states associated with acid hypersecretion.
2. What are the three preoperative criteria for making a diagnosis of ZES?
3. List five indications for obtaining a fasting serum gastrin level.
4. In assessing efficacy of medical treatment in ZES, what procedure should guide selection of drug dose and dosing interval?
5. What are the four major current indications for total gastrecomy in the treatment of ZES?

(Answers on page 35)

Suggested Readings

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THE CASE OF THE JUMBLED *JOURNAL*

A few copies of the December issue of the *Journal* were improperly assembled. This resulted in duplication of some pages and omission of others.

Please check your December issue; if yours is one of the aberrant ones, please contact the KMS office for a replacement copy.



Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Insulin

Question: I have heard that there are many "purified" insulins now available. Would you provide me with information about these new products and which ones are currently available in the United States?

Answer: Currently, there are more than two dozen insulin products approved by the Food and Drug Administration for marketing in the United States. These products differ in purity levels and specie source, two important factors in the clinical use of insulins. However, inconsistent terminology describing purity levels has led to some confusion to both the physician and the patient.

Most commercially available insulins contain a mixture of both pork and beef insulin. The pork insulin has been selected for further purification processes since it is closer to human insulin and believed to be less immunogenic. Greater than 80 per cent of diabetic patients with systemic or persistent local allergy to insulin will tolerate pork insulin better than beef insulin.[1]

Regardless of specie source, insulin contains various proinsulin and noninsulin proteins. These "contaminants" are measured in parts per million (ppm). Since the introduction of the purified insulins, the USP has designated three classes of insulin based on purity. These are (1) Conventional or the pre-1972 insulins (>10,000 ppm); (2) Single Peak (<50 ppm); and (3) Single Component (<10 ppm). When a patient has been stabilized on a large dosage of conventional insulin and is changed to a purified insulin, reduction of dosage may be necessary. Although manufacturers recommend a 20 per cent reduction in dose, decreases of 9-36 per cent have been reported in the literature.[2-5] It should be noted that only a small number of patients may require this dosage reduction. Unfortunately, it is not possible to identify beforehand which patients require dosage alterations. If adjustment is needed, it may occur with the first dose or over a period of several weeks.

Candidates identified for purified insulins are patients with (1) chronic lipoatrophy; (2) chronic insulin allergy; and (3) insulin resistance. These three groups comprise less than 5 per cent of all diabetic patients. Some evidence suggests that lipoatrophy is more common with less pure insulins.[6] However, there is limited evidence that purified insulins may be useful in prevention or treatment of other complications of insulin therapy. Although the purified insulins are less immunogenic than the previous insulins, the long term advantages remain to be established.

INSULINS CURRENTLY AVAILABLE IN THE U.S.

PURITY	SPECIE SOURCE	TYPE OF ACTION		
		RAPID ACTING	INTERMEDIATE ACTING	LONG ACTING
< 10 PPM	PORK	ILETIN II REGULAR (LILLY) †REGULAR (SQUIBB) ACTRAPID (NOVO) SEMITARD (NOVO) QUICK (NORDISK) MIXTARD -30% REG/70% NPH (NORDISK)	ILETIN II-NPH (LILLY) ILETIN II-LENTE (LILLY) MONOTARD (NOVO) NPH (NORDISK) PROTAPHANE-NPH (NOVO)	ILETIN II-PZI(LILLY)
	BEEF	ILETIN II-REGULAR (LILLY)	ILETIN II-NPH (LILLY) ILETIN II-LENTE (LILLY) †NPH (SQUIBB) †LENTE (SQUIBB)	ILETIN II-PZI (LILLY) ULTRATARD (NOVO)
	BEEF-PORK		LENTARD (NOVO)	
< 25 PPM	PORK	††REGULAR (SQUIBB)		
	BEEF	††SEMILENTE (SQUIBB)	††LENTE (SQUIBB) ††NPH (SQUIBB)	††ULTRALENTE (SQUIBB)
< 50 PPM	BEEF-PORK	ILETIN-REGULAR (LILLY) ILETIN-SEMILENTE (LILLY)	ILETIN-NPH (LILLY) ILETIN-LENTE (LILLY)	ILETIN-PZI (LILLY) ILETIN-ULTRALENTE(CL)

†WILL BE AVAILABLE IN EARLY 1982
††WILL REPLACE PREVIOUS "CONVENTIONAL" INSULINS (>10,000PPM) MANUFACTURED BY SQUIBB

Submitted by
Joyce Generali, R.Ph.

References available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

Abstracts

Presented at the 1981 Annual Meeting, Kansas Chapter, American College of Physicians

Percutaneous Transluminal Coronary Angioplasty: Preliminary Results

Joseph P. Galichia, M.D.; Roger W. Roberts, D.O.; Gregory F. Duick, M.D.; Thomas A. Wiberg, M.D.; Richard A. Steckley, M.D. and Nancy Nelson, R.N., Wichita

Percutaneous balloon dilatation was carried out in ten patients with high grade stenoses involving a single coronary artery. All of these patients had angina pectoris, which was unsuccessfully managed with medical therapy, and were thereby surgical candidates. Three patients had unstable angina and did not undergo preoperative stress testing. Seven patients had positive treadmill tests, and all of these patients had a one month or longer trial of medical management. Mean luminal diameter narrowing decreased from 86% ± 12.9% (SD) preangioplasty to 31% ± 15.5% (SD) post dilatation (p less than .001). Ninety per cent of lesions were successfully dilated. Dilatations of five high grade stenotic lesions of the right coronary artery were attempted successfully. Of dilatations attempted in five patients with left anterior descending lesions, four were successful. In the one unsuccessful attempt, a subtotal occlusion of the left anterior descending artery could not be crossed. No complications were incurred in this group of patients. Relief of symptoms and return to asymptomatic status was achieved in all patients who underwent successful dilatation. Five of seven patients had repeat treadmill tests. All five had increased exercise tolerance and negative treadmill tests post angioplasty. The one patient in whom dilatation could not be carried out underwent immediate bypass surgery with good surgical results. In conclusion, balloon dilatation is an effective alternative to surgical management in patients with high grade proximal single vessel coronary disease.

Usefulness of Two-Dimensional Echocardiography in Infective Endocarditis

George Gura, M.D.; Fred Nasser, M.D.; Abdul Tajik, M.D. and James Seward, M.D., Rochester, Minnesota

Detection of vegetations by M-mode echocardiography has been of major importance in the early diagnosis and management of patients with infective endocarditis (IE). Wide angle two-dimensional echocardiography (2-D) represents a major technological advance because of the ability to image the heart more completely and to obtain images that are spatially correct and resemble familiar cardiac anatomy.

This study was an attempt to evaluate the usefulness of 2-D in 75 patients with proven IE on native valves. The patients were divided into three groups: Group I (Gp I) with definite vegetations — 39 (51%); Group II (Gp II) with probable vegetations — eight (12%); and Group III (Gp III) with no vegetations — 28 (37%). Direct visualization of the valves at surgery or autopsy was possible in 29 patients. Vegetations were confirmed in 26 of 29 patients (90%).

Gp I patients had: (1) higher mortality rate (7/39 — 18%) than Gp II (0/8 — 0%) or Gp III (2/28 — 7%); (2) a higher incidence of surgical intervention (16/39 — 41%) than Gp II (0/8 — 0%) or Gp III (1/28 — 4%); and (3) a higher incidence of embolization (20/39 — 51%) than Gp II (2/8 — 25%) or Gp III (3/28 — 11%). Complications such as annular or myocardial abscess formation (five patients), or valvular disruption were identified. We concluded that 2-D has major diagnostic and prognostic application in the detection of vegetations and the identification of patients at high risk of complication and in need of surgical intervention.

A Proposed Explanation for the Development of ST Segment Depression After Exercise

Ann Allegre, M.D. and Marvin Dunn, M.D., Kansas City, Kansas

An unexplained phenomenon in treadmill exercise testing is the development of ST segment depression after exercise is completed. Data was collected from the five patients who have exhibited this phenomenon in our series of treadmill tests done for coronary artery disease in the past six months. Of these five patients, four developed the ST segment depression just after moving from a sitting to a supine position, which is routinely done two minutes after exercise is completed. Three of the patients had a rise in diastolic blood pressure of 10-15 mmHg just after becoming supine. Only one patient complained of chest pain at this time. Pulse rate was gradually decreasing during the recovery period. It is postulated that the postural rise in blood pressure is an important factor in producing the delayed ST segment depression in some patients.

Spontaneous Conversion of Long-Standing Atrial Fibrillation

Douglas Gardner, M.D. and Marvin Dunn, M.D., Kansas City, Kansas

Spontaneous conversion to sinus rhythm after prolonged atrial fibrillation is an uncommon occurrence with fewer than 20 cases described in the literature. We recently encountered three patients with spontaneous conversion to sinus rhythm after the presence of atrial fibrillation for periods of 9-13 years.

A 50-year-old white female with a history of rheumatic heart disease underwent mitral commissurotomy in 1956. She developed atrial fibrillation in 1971 and spontaneously converted to sinus rhythm in 1979.

A 52-year-old white female with rheumatic heart disease and a porcine mitral heterograft developed atrial fibrillation in 1966 and spontaneously converted to normal sinus rhythm in 1979.

A 62-year-old male with rheumatic heart disease developed atrial fibrillation in 1971 and was found to be in atrial tachycardia with 2:1 block in 1980.

No patient was digitalis intoxicated. One had a prolonged P-R interval. Echocardiography showed poor to absent left atrial contraction.

Spontaneous conversion from atrial fibrillation occurs for reasons that are unclear.

Detection of Deep Vein Thrombosis in Postoperative Coronary Bypass Patients: An Evaluation of Clinical, Invasive, and Noninvasive Methods

G. William Pogson, M.D.; Gerald S. Weinstein, M.D. Ben D. McCallister, M.D.; James J. Harbrecht, M.D.; G. David Dixon, M.D. and William R. Hamaker, M.D., Kansas City, Missouri

Deep vein thrombosis (DVT) is a major cause of morbidity in patients (pts) following coronary artery bypass (CAB). To study methods of detecting DVT, 200 consecutive postoperative CAB pts were examined daily. A randomly selected sub-group of 78 asymptomatic (asx) pts had impedance plethysmography (IPG) and Doppler ultrasound (DU) on postoperative day six or seven. Pts with abnormal IPG or DU (9 pts) or clinical signs of DVT (46 pts) underwent venography (VG). Thirty-six of 46 pts with calf tenderness or leg swelling (LS) had IPG and DU within 48 hours of VG. Thirty-three of the 200 pts had DVT documented by VG (16%). CT was a stronger indicator of DVT (24/26 pts — 92%) than painless LS (5/20 — 25%). A positive VG occurred in one of nine (11%) asx legs opposite to those with LS. IPG and DU had accuracies of 59 and 58 per cent respectively. DVT was not significantly related to age, postoperative ejection fraction, number of vein grafts, or obesity.

Conclusion: DVT occurs in at least 16 per cent of pts following CAB. Although LS, IPG, and DU were not helpful or

reliable indicators of DVT, CT was an accurate indicator. Therefore, careful attention to clinical CT is important in the detection of DVT and is superior to combined IPG and DU in pts following CAB.

Late Rupture of a Left Ventricular Aneurysm

Robert E. Roeder, M.D.; John Long, M.D. and Douglas A. Marples, A.S.C.P., Topeka

A 42-year-old man suffered an anterolateral myocardial infarction, complicated by pericarditis and the development of a left ventricular aneurysm. Left ventriculography was performed six weeks after the initial event, revealing a large apical aneurysm. As the lesion was not producing symptomatic hemodynamic compromise, he was discharged on anticoagulants. Ten weeks following discharge the patient had the abrupt onset of cardiogenic shock. Cardiac enzymes were not elevated. Repeat left ventriculography revealed further enlargement of the left ventricular aneurysm.

At thoracotomy, findings of rupture of a true left ventricular aneurysm were present. Complete hemopericardium with cardiac tamponade had been prevented by pericardial adhesions surrounding the site of rupture. Successful aneurysmectomy was performed, and the patient's recovery was uneventful.

Congenital Pulmonary Arteriovenous Fistulas: Clinical and Angiographic Findings

Roger J. Dreiling, M.D.; Floyd Atkins, M.D.; Joseph Chang, M.D. and David Pugh, M.D., Kansas City, Kansas

Congenital pulmonary arteriovenous fistulas (PAF) are rare, and their natural history is incompletely understood. They may be associated with arteriovenous fistulas involving skin, mucous membranes, and sometimes other organs such as the liver.

In the last ten years, four cases of congenital PAF have been studied at the University of Kansas Medical Center. Common presenting manifestations were peripheral telangiectasias, digital clubbing with cyanosis, and dyspnea on exertion. Complications resulting from PAF were polycythemia with cerebrovascular accident in one patient; intrapleural hemorrhage in one patient; and frontal lobe anerobic brain abscess in one patient. Two patients had hereditary telangiectasia with frequent epistaxis, and one of our patients manifested hemoptysis prior to diagnosis. Laboratory data showed polycythemia in two of the four patients, and these patients also had systemic arterial oxygen desaturation. All patients had abnormal chest x-rays with solitary or multiple pulmonary nodules. At pulmonary angiography, two patients were found to have large peripheral aneurysmal dilatation of the PAF, while two other patients exhibited multiple, small, peripheral PAFs. One patient also had arteriovenous malformations of the liver confirmed at the time of abdominal angiography. The diagnosis of pulmonary arteriovenous fistula should be suspected in patients exhibiting the clinical triad of digital clubbing, cyanosis, and dyspnea on exertion in association with solitary or multiple pulmonary nodules.

Diffuse Intrapulmonary Hemorrhage Without Immune Complexes or ABMA Treated With Plasmapheresis, Prednisone, and Cytoxan: Presentation of Two Cases and a Review of the Literature

Stephanie Nellis, M.D.; Martin Welch, M.D. and W. Tom Meredith, M.D., Wichita

Diffuse intrapulmonary hemorrhage has been classified into three categories based on immunopathologic studies. These include one in which anti-basement membrane antibody (ABMA) is present, one with immune complexes, and a third with evidence of neither by immunofluorescence or electron microscopy (EM). Two patients are presented who fit into the third category. *Case one* is a 46-year-old male with a six-month history of pulmonary hemorrhage and anemia. Serum ABMA was negative. Lung biopsy showed hemosiderin laden histiocytes without vasculitis. Immunofluorescence failed to show deposition of immunoglobulins, C3 or ABMA, and EM revealed no electron dense deposits suggesting immunoglobulins. *Case two* is a 63-year-old male with pulmonary hemorrhage and renal failure. The immunologic survey and serum ABMA were negative. The lung biopsy findings were similar to Case one, and the kidney biopsy showed crescentic glomerulonephritis. Both cases were successfully treated with plasmapheresis, prednisone, and Cytoxan.

A review of the English language literature, since the four cases with negative EM and immunofluorescence cited in the above 1975 classification, supports the existence of category three of diffuse intrapulmonary hemorrhage. There are five reported cases with both negative EM and immunofluorescence, and three cases with only immunofluorescence being done and reported as negative. There was one case with only EM studies, which showed no electron dense deposits. The patients presented in this report are offered in support of the existence of a category of diffuse intrapulmonary hemorrhage without an immunologic etiology.

Early Bronchoscopy in the Burn Patient

Michael J. Waxman, M.D.; Gerald R. Kerby, M.D. and Mani M. Mani, M.D., Kansas City, Kansas

Thermal injury and smoke inhalation can result in upper airway obstruction, disturbance of the mucociliary clearance mechanism with tracheobronchitis, and more distal injury resulting in acute pulmonary edema. A review is made of the bronchoscopic findings of 67 patients admitted to the Burn Unit at the University of Kansas Medical Center between 1976-1980. Bronchoscopy was performed on all patients within 24 hours of admission. On the basis of early bronchoscopic findings, elective intubation was performed on 33 of the 67 patients.

Death secondary to acute upper airway edema can be prevented by early intubation. Early bronchoscopy can predict possible obstruction as well as identify involvement of the lower airway by heat and smoke. Pulmonary morbidity and mortality in the early phase of the burn patient are compared to previous data. The efficacy of early bronchoscopy and intubation in the burn patient will be evaluated.

Hepatitis A With Associated Parkinsonian-Like Syndrome

Kim Arganbright, M.D. and Lew W. Purinton, M.D., Wichita

A 30-year-old white female with a 36-hour history of nausea, vomiting, and increased temperature had decreased oral intake and became quite lethargic. Admission day temperature was 39.5-41.0C, and was associated with an erythematous rash on the entire body. Results of physical examination were within normal limits, except for temperature of 36.8C. Urine, vaginal, cervical, blood ($\times 3$), and throat cultures yielded negative results. White blood count was 3.3; H&H, 14.6-43.7 (normal differential); SGOT, 1375; SGPT, 1635; LDH, 945; alkaline phosphatase, 231; and total bilirubin, 1.3. Diagnosis of hepatitis was made. The patient had been traveling prior to illness, but had no known exposure. Hepatitis B surface antigen, hepatitis B core antigen, and hepatitis B core antibody were negative; hepatitis A immunoglobulin antibody was positive. Nausea and vomiting continued during the first two to three days of hospitalization. Liver enzymes continued to increase (on August 19, SGOT was 4630; SGPT, 8000 and alkaline phosphatase, 305); total bilirubin increased from 4.1 to 9.6; and blood ammonia fluctuated between 55 and 65 from August 19-24. Prothrombin

time increased to 16 seconds with control of 11 on August 23 and 26. On August 27, SGOT was 154; SGPT, 620; alkaline phosphatase, 275; and bilirubin, 6.7. The patient became restless and tremulous, and manifested muscle rigidity. She became almost catatonic and began manifesting inappropriate behavior. She paced the floor 24 hrs/day and began to show an increase in involuntary tremors; she had a slow shuffling gait, masked facies, bradykinesia, and other symptoms of a Parkinsonian-like syndrome. A phantom pillow sign was noted. A neurologic consultant felt she had Von Economo's syndrome — a post infectious Parkinsonism from a viral infection. Spinal fluid was normal. On August 28, severe swallowing problems developed and she was unable to take medicine or food, or to handle her own secretions. A Dobhoff feeding tube was placed in the stomach. Following the institution of enteral feeding, Benadryl, Artane and intravenous Decadron, her condition improved. Although still recovering, she is markedly improved.

Experience With Flexible Sigmoidoscopy in a Community Hospital

M. Ashraf Sufi, M.D., Topeka

Flexible sigmoidoscopy was compared with rigid sigmoidoscopy for length of colon examined, patient discomfort, time required, and diagnostic yield.

Two hundred fifteen patients were examined, 115 by rigid sigmoidoscopy and 100 by 68 cm long single-channel Olympus flexible sigmoidoscope. Eighty per cent of patients were symptomatic (abdominal pain, diarrhea, constipation, and rectal bleeding); 20 per cent were asymptomatic. Sixty per cent were male and 40 per cent were female. Age range was 40-80 years. No sedation was given, and preparation was identical in both procedures. Informed consent was obtained. All procedures were performed by the same gastroenterologist.

Average length of colon examined with flexible sigmoidoscopy was 50 cm compared to 19 cm with the rigid sigmoidoscope. Average time required for flexible sigmoidoscopy was ten minutes compared to six minutes for rigid sigmoidoscopy. Patient discomfort was minimal with the flexible sigmoidoscope, moderate with the rigid sigmoidoscope.

Fourteen of 100 patients had recto-colic lesion found by flexible sigmoidoscope. Twelve were found to have 14 polyps. Four were hyperplastic; nine were adenomatous, and one was villous adenoma. Four of these polyps were greater than 20 cm from anal verge. Three polyps had malignancy at the head.

Eight of 115 (7%) of patients had recto-colic lesions found by rigid sigmoidoscope. Six were polyps; four were adenomatous and two were hyperplastic. One patient had ulcerative colitis and one had cancer of the rectum.

Conclusion: This study indicates that flexible sigmoidoscopy is superior to rigid sigmoidoscopy for diagnostic yield, patient discomfort, and area of colon examined.

Multiple Amoebic Liver Abscesses in a Kansas Traveler

J. Barker, M.D.; D. Hinthorn, M.D.; D. Preston, M.D.; E. Levine, M.D. and C. Liu, M.D., Kansas City, Kansas

A 43-year-old man vacationed in Ixtapa, Mexico, in August 1979. After returning, he had nausea, vomiting, and diarrhea for three days which subsided without treatment. One month later, he developed fever and right shoulder pain. On admission, he was jaundiced and acutely ill. He had a right pleural effusion and an enlarged tender liver. A CAT scan demonstrated multiple abscesses in both lobes of the liver. Fluid obtained by needle aspiration was culturally sterile, and no amoebae were seen. Bilirubin was 13; SGOT, 391; and cholesterol, 19. Therapy was initiated with intravenous gentamicin, chloramphenicol, and oral metronidazole. Transient ascites developed, but resolved without surgical intervention. The patient had a stormy clinical course and was transferred to KUMC. Definitive diagnosis was established by repeatedly negative serology to hepatitis A and B, but rise of antibody from 1 : 128 to 1 : 1024 by indirect hemagglutination test for *Entamoeba histolytica*.

Contrary to statements in the literature, this patient with amebiasis exhibited multiple hepatic abscesses, involving left as well as right lobes, and severe liver dysfunction. Diagnosis is best made from clinical history and examination of aspirated fluid for amoeba. Rise of amoebic antibody titers were diagnostic but delayed. Amoebic liver abscess responds well to metronidazole therapy. In contrast, all pyogenic liver abscesses must be drained surgically.

The CAT scan was of value in this case in making the initial diagnosis, in guiding needle aspiration, and in demonstrating delay in resolution.

Amoebic liver abscess is rare in Kansas. With the increase in overseas travel, and the recent influx of immigrants, we should expect amebiasis to be a consideration more often in the future.

Bone Marrow Transplantation in Treatment of Acute Leukemia

David Bodensteiner, M.D.; Mammo Amare, M.D. and Barry Skikne, M.D., Kansas City, Kansas

Between January and August 1980, four patients with acute leukemia [three with myeloblastic leukemia in relapse and one with chronic granulocytic leukemia (CGL) in blast crisis] received bone marrow transplants from their HLA-MLC identical sibling donors, after preparation with high-dose cyclophosphamide and total body irradiation. One patient (CGL in blast crisis) died of sepsis without evidence of engraftment 16 days after transplantation. Three had successful engraftment with sustained marrow function. One patient is now six months post-transplant and is entirely well; one has mild liver dysfunction but is well 3½ months post-transplantation; and the third has acute graft-versus-host disease, 1½ months after transplantation. While no firm conclusion can be drawn, our limited experience with bone marrow transplantation (BMT) in end-stage leukemia compares favorably with larger series in which the long-term survival rate is 15-25 per cent. Although BMT has a limited role in advanced-stage leukemia, recent studies have shown that, if done early in the course of the disease, it may substantially improve the long-term survival rate (62-63%), offering cure to the majority of patients. Thus BMT should be considered for all patients with acute nonlymphocytic leukemia in first remission and with acute lymphocytic leukemia in second remission.

Hypercalcemia With Encephalopathy, Non-Oliguric Renal Failure and Extensive Metastatic Calcification in a B-cell Non-Hodgkin's Lymphoma

Ming-S. Lok, M.D.; Carol J. Trautt, M.T., A.S.C.P., Albert K. Kim, M.D. and K. Paul Poulose, M.D., Leavenworth

A patient with blastemia presented with hepatosplenomegaly, hypercalcemic encephalopathy, and non-oliguric renal failure. Light microscopic morphology and cytochemical staining suggested that the immature cells were prolymphocytes and lymphoblasts (transformed lymphoid cells). The presence of monoclonal surface and cytoplasmic immunoglobulin (u, r and k type) confirmed the B-cell origin of the malignancy. The possibility of hairy cell leukemia was excluded by the tartrate-resistent acid phosphatase study and in vitro phagocytosis test. Autopsy showed massive and diffuse involvement of the spleen, focal and patchy disease in the marrow, and absence of gross lymphadenopathy. These findings indicate that the spleen was the primary site of the lymphomatous process. Extensive metastatic calcification involving many organs and the forma-

tion of numerous sandy calculi in the kidneys and bladder were also noted. The ectopic production of parathyroid hormone is probably the pathogenic mechanism of the deranged calcium homeostasis.

Mu Heavy Chain Disease: Report of the 23rd Case

Pamela S. Maben, M.D. and Harry E. Hynes, M.D., Wichita

Mu heavy chain disease is a rare disorder associated with lymphoid malignancies. Patients have a defect in the heavy chain of their IgM proteins. The diagnosis is made by finding a rapidly migrating protein which reacts only with antisera to the heavy chains of IgM and not with antisera to light chains by immunoelectrophoresis and immunodiffusion.

Our patient presented with symptomatic hypercalcemia one year after his chronic lymphocytic leukemia and mu heavy chain disease was discovered. He had Bence-Jones protein in his serum and urine, the rapidly migrating IgM heavy chain in his serum, vacuolated plasma cells in his bone marrow, peripheral lymphadenopathy, and splenomegaly. Lymph node biopsy showed immunoblastic sarcoma, which is a particularly virulent lymphoma. Parathormone level tests were done at two different institutions; one level was normal, the other very high suggesting ectopic hormone production. The patient responded initially to combination chemotherapy, hydration, and diuretics. The patient had two further episodes of hypercalcemia correlated with worsening of his lymphoma before he succumbed to pneumonia.

Our patient was similar to the first 22 cases of mu heavy chain disease except for his hypercalcemia. The other 22 cases had various survival rates; from more than ten years to months. Survival seemed to correlate with the patient's lymphoid malignancy and not with the level of mu heavy chain protein. Structural studies have been performed on several mu heavy chain disease proteins showing deletions in the Fd portion of the heavy chain. Structural studies are pending on the mu heavy chain protein of our patient.

Red Cell Antibody Detection by the ELISA

David Bodensteiner, M.D.; Barry Skikne, M.D.; Peggy Brown, M.A., M.T., A.S.C.P., S.B.B. and Fred Plapp, M.D., Kansas City, Kansas

The enzyme-linked immunosorbent assay (ELISA) was employed in the study of red cells from patients with autoimmune hemolytic anemia (AIHA). The ELISA uses an anti-IgG conjugated to alkaline phosphatase to determine the number of IgG molecules per red cell. Normal values are less than 50 IgG molecules/RBC. We have studied six AIHA patients — four Coombs positive and two Coombs negative. In the four Coombs positive patients, the IgG level per RBC ranged from 1,000 to greater than 7,000, while in the Coombs negative patients the IgG level was elevated to 650 in both. Four of these patients — two Coombs positive and two Coombs negative — are in clinical remission, and the red cell IgG level has dropped to normal. The remaining two have continuing clinical and laboratory evidence of hemolysis, and the IgG level has remained elevated. We have found the red cell ELISA helpful in diagnosing and following the clinical course of patients with AIHA. This is particularly true in the Coombs negative group since the ELISA is more sensitive and can detect smaller increases in red cell IgG than required for a positive Coombs.

Expanded Use of Discriminant Functions in Evaluation of Red Cell Microcytosis

Ming-S. Lok, M.D. and Carol J. Trautt, M.T., A.S.C.P., Leavenworth

A linear discriminant function (DF′ = MCV − 5 × Hb − RBC − 3.4), two ratio discriminant functions (MCV/RBC and MCH/RBC), and the erythrocyte count (millions/μl) were calculated from routine red cell profiles in 78 consecutive adult patients having mean corpuscular volume (MCV) of less than 80 fl. Eight patients (group A) had negative values of DF′, MCV/RBC ratio below 13, MCH/RBC ratio below 3.8, and/or erythrocyte count above 5 million/μl. Study of this group of patients showed that three had B-thalassemia minor, two had probable L-thalassemia minor, one had hemoglobin C trait, and two had polycythemia vera with incidental iron deficiency. Seventy patients (group B) had positive values of DF′ and MCV/RBC ratio above 13. This group consisted of 65 patients with iron deficiency, three patients with anemia of inflammatory disorders, and two patients with sideroblastic anemia. The usefulness of expanding the application of these simple calculations to detect thalassemia trait and evaluate other causes of red cell microcytosis is discussed, and a scheme of work-up strategy is proposed.

Malignant Plasmacytic-Lymphocytic Lymphoma: Demonstration of a Serum Inhibitor of Myelopoiesis and Response to Combination Chemotherapy

Larry Balentine, M.D.; Barry Skikne, M.D.; Chan Park, M.D. and Sean Lynch, M.D., Kansas City, Kansas

A case of malignant plasmacytic-lymphocytic lymphoma with associated leukopenia is described. The colony-forming unit (CFU-C) assay was used to quantitate stem cells and test for the presence of suppressor cells and/or serum factors inhibiting bone marrow differentiation. Significant suppressor cell activity was not demonstrated. The pre-treatment serum, when cocultured with normal bone marrow cells, produced a significant decrease in CFU-Cs. Post treatment serum under the same conditions did not result in a reduction in CFU-Cs. Treatment with combination chemotherapy (prednisone and cyclophosphamide) produced a clinical response with correction of the leukopenia.

Membranoproliferative Glomerulonephropathy Complicating Immunologic Diseases

Mary Nielsen, M.D. and Hugo Weber, Jr., M.D., Wichita

Membranoproliferative glomerulonephropathy (MPGN) denotes a group of disorders that are idiopathic or may be associated with primary illness. Histologic features of type I (subendothelial deposits) and type II (dense deposits) MPGN will be illustrated in three patients with idiopathic disease. Two patients with MPGN complicating immunologic disease will be discussed — one episode complicating rheumatic disease, the other a B cell dyscrasia.

A 61-year-old female was seen for a six-month complaint of recurrent bilateral ankle inflammation. Proteinuria (0.6 gm/24 hrs) and microscopic hematuria were present. Immunologic studies yielded the following results: complement profiles demonstrating classic pathway activation; ANA, neg × 3; anti-DNA Ab absent; RA titer, 1:320; serum cryoglobulin, absent. Serum creatinine was 1.0 mg/100 ml and creatinine clearance, 76 ml/min. Percutaneous renal biopsy demonstrated typical features of MPGN with mesangial proliferation, subendothelial extension of mesangial cytoplasm into capillary walls, and mesangial and subendothelial capillary electron dense deposits.

A 53-year-old male presented with proteinuria (12.7 gm/24 hrs) and hematuria. Serum and urine protein electrophoresis identified an IgG lambda monoclonal gammopathy. Serum creatinine was 12.7 gm/100 ml, and creatinine clearance was 12 ml/min. Renal biopsy demonstrated mesangial interpositioning and subendothelial deposits characteristic of MPGN. Four courses of treatment with vincristine, melphalan, Cytoxan, and prednisone were given.

Increasing use of renal biopsy has allowed identification of MPGH as a complication of immunologic diseases.

Thyroid Antigen-Antibody Immune Complex Glomerulonephropathy

Hugo Weber, Jr., M.D., and Leo Cawley, M.D., Wichita

The rare occurrence of thyroid antigen-antibody immune complex mediated focal membranous glomerulonephropathy is described in a 23-year-old female with thyroiditis and proteinuria.

The thyroiditis was initially characterized by an enlarged thyroid gland and abnormal function studies (TSH, 95 μμ/ml; T-4, 3 μg/100 ml; T-3, 70%; index, 4.2). Antibodies to thyroid antigens were detected (anti-thyroglobulin, 1:40, 960; anti-microsomal, 1:160). Twenty-four-hour quantitative protein excretion was 1.6 gm, and creatinine clearance 90 ml/min.

Percutaneous renal biopsy showed several sclerotic glomeruli. Silver staining demonstrated the characteristic spikes of membranous glomerulonephropathy extending off the basement membranes of the peripheral capillary loops. Capillary walls were thickened. Electron microscopy confirmed the presence of focal, principally epimembranous, deposition of electron dense deposits. Direct immunofluorescent studies revealed 2 + IGG, 3 − C3, ± IGA, and ± fibrin granular deposits along the capillary loops.

Localization of thyroglobulin was carried out by indirect immunoperoxidase microscopy on paraffin imbedded biopsy tissue.

Fenoprofen (F) Induced Acute Renal Failure Presenting as Rapidly Progressive Glomerulonephritis

D. Artzer, M.D. and D. Diederich, M.D., Kansas City, Kansas

A 64-year-old man presented with a recurrence of nephrotic syndrome (NS) associated with rapidly progressive azotemia (AZ) and oliguria. Three months earlier the patient developed an insidious NS (24-hr urinary protein, 29 gm). Evaluation revealed AZ (Cr 3.1) and stage I renal cell carcinoma. Spontaneous remission of AZ and NS were noted prior to nephrectomy. One month after admission Cr was 1.5 and total protein excretion, 77 mg. Recurrence of NS was noted two months later accompanied by progressive, severe AZ (Cr, 11 mg/100 ml) and oliguria. Red cell casts and lipiduria were present. Mechanical causes of NS and AZ were excluded. There was no evidence of tumor recurrence. An acute interstitial nephritis with normal glomeruli noted on renal biopsy prompted review of medication intake. Intermittent F usage over a period of two years was elicited.

Upon discontinuance of F, the NS and AZ spontaneously remitted on both occasions without the need for dialysis or immunosuppression.

Analysis of the Microenvironment of a Human Polycystic Kidney

S. Schwab, M.D.; A. Gobuty, Pharm.D.; F. F. Cuppage, M.D.; D. Hinthorn, M.D. and J. Grantham, M.D., Kansas City

Polycystic kidney disease is common in the United States affecting one person in 500. Infection of these cysts is a common clinical occurrence. Little is known of the internal environment of these cysts, including antibiotic penetration. We studied a patient with polycystic kidney disease who had an apparent renal infection with negative urine and blood cultures. Despite therapy with parenteral clindamycin and gentamicin, he continued to have continuous back pain and persistent fever for which unilateral nephrectomy was performed. Fluid from 22 cysts was aspirated immediately after nephrectomy and analyzed for PO_2, PCO_2, pH, and antibiotic levels. The fluid was cultured for aerobes, anaerobes, viruses and chlamydia.

PO_2 was less than 50 mmHg in seven cysts (0-47 mmHg). Abundant oval fat bodies were present in these cysts and probably accounted for the thick consistency, dark color, and low PO_2. Clindamycin and gentamicin were present in all cysts analyzed. Serum level of clindamycin was 4.0 μg/ml with a mean cyst concentration of 0.3 μg/ml ± 8.5 μg/ml. Gentamicin serum trough level was 1.9 μg/ml with a mean cyst concentration of 1.0 μg/ml ± 0.5 μg/ml. All cultures for bacteria, viruses, and chlamydia were negative.

We postulate that RBC breakdown in pigmented cysts forms oval fat bodies, consumes O_2, and creates an hypoxic environment. Hypoxia could allow colonization with aerobic or anaerobic bacteria with subsequent infection. Clindamycin levels in cysts were in the therapeutic range while gentamicin levels were suboptimal for many organisms that cause urinary tract infections.

Severe Hyponatremia Due to Psychogenic Polydipsia

Ganjur H. Guruprakash, M.D.; H. Lok, M.D. and K. P. Poulose, M.D., Leavenworth

Water intoxication has been reported in schizophrenics with compulsive polydipsia. Many of these patients have associated syndrome of inappropriate antidiuretic hormone secretion (SIADH). We report a case of severe hyponatremia in a patient with compulsive polydipsia who showed no evidence of SIADH.

A 59-year-old white male was drinking large amounts of water to "flush a toothbrush stuck in the throat." He had a several year history of schizophrenia and hypertension. He had experienced two episodes of severe hyponatremia, once resulting in seizures and another time in a stuporous state. During both of these episodes the patient was taking haloperidol and diuretics, which could have been a contributing cause of severe hyponatremia. These medications were discontinued for three weeks, and the patient was reevaluated.

Blood sugar, BUN, creatinine, and urine were normal. Pertinent serum electrolytes, urine electrolytes, and osmolality values are as follows:

Date	*Na*	*K*	*Cl*	*Co2*	*U.Na*	*U.Osm*	*Symptoms*
6/21	114	3.7	77	29.7	—	—	Stupor
6/30	102	2.5	59	33.3	—	—	Seizures
7/25	119	5.2	86	22.1	4	60	Confusion
7/29	123	4.3	81	27.7	6	106	—
8/01	121	4.0	81	30.0	6	86	—
8/06	132	4.8	94	26.7	12	108	—

Recorded output, which was incomplete because of lack of co-operation from the patient, varied between 8-18 L/day. At no time was urine osmolality inappropriately high, excluding the possibility of SIADH.

In our patient episodes of severe hyponatremia improved without any specific treatment. However, fatal cases have occurred which indicates the need for vigorous management of severe hyponatremia due to water intoxication.

Eaton-Lambert Syndrome: Unusual Presentation of Prostatic Cancer

Ganjur H. Guruprakash, M.D. and Iftekhar Ahmed, M.D., Leavenworth

Neurologic complications of carcinoma of the prostate (CP) are generally due to metastatic disease. There are occasional reports of non-metastatic neurologic complications. We report a case of CP associated with Eaton-Lambert syndrome (ELS).

A 59-year-old man was admitted with a history of dysphagia and weight loss. He also complained of nocturia and dribbling of urine. Examination revealed a mass 6 x 4 cms involving the right clavicle. Prostate was enlarged and hard in consistency. Results

(Continued on page 30)

The President's Message



This day and age, anticipated to be the age of enlightenment, has developed into an age of utter confusion — at least when we speak of medical information to the public. Every day people hear and see conflicting reports regarding the causes and cures of cancer, the efficacy (or lack thereof) of vitamin C, special exercises, cholesterol, acupuncture, reflexology, manipulations, prolotherapy, holotherapy, aloe vera, "painless" weight reducing diets, and new remedies for almost any malady — just to name a few.

The medical profession is not entirely without blame for this prevailing situation. For example, take the case of research papers. Results are often released too early — prior to verification by other investigators, before sampling is statistically adequate, or before all variables are properly evaluated. Later, as we so often see, all the excitement is found to be unwarranted. Undoubtedly, the reasons for this premature reporting are many, among them eagerness for personal renoun; the intense desire by publishers of periodicals to be "the first"; sensationalism (the *National Enquirer* type of mentality); and the need to keep research grants coming in.

Loss of credibility is inevitable. The public is beginning to think that the medical profession "doesn't know what it's talking about." As confusion becomes rampant, the public's confidence in medical advice will lessen, not only in what comes across the news media, but also, unfortunately, in the one-to-one setting between doctor and patient, with the result that more people will decide for themselves what is good advice and what is not. The charlatans and the super advertisers will then have their heyday.

It has been recently said that in the next few years, our patients will be our greatest competitors. This may be beginning to happen.

Fraternally,

Herman W. Kiesterman M.D.

President



PACing Them In

The technologic and pharmacologic "revolutions" in medicine of recent decades have obscured a different type of change in the physiology of the medical body — its increasing political function. Physicians generally haven't taken too readily to politics — "political" politics, that is — identifying the whole activity with its seamier aspects or holding to a certainty that no good was likely to come from it. Distilled down, this attitude may stem from the fundamental dedication to autonomy that physicians have exercised in their relationship with patients, making them reluctant to aspire to political activism since this might lead to the relinquishment of some control to the opinions or decisions of others. Certainly the active participants in the political process have far more often come from other areas — business, labor, and above all, law. Physicians have had only peripheral contact, usually in consulting capacities when some distinctly medical measure was at hand and, of course, at times of raising funds (in which physicians have been known to the pros as consistently penurious, enough in itself to minimize their political clout). This is not to discount the occasional physicians, past and current, who have entered the political lists to their considerable credit but they stand out partly because they are the exception.

Times do change, however. There can be no question but that this century has seen the medical world subjected to a spate of political pressures never before experienced. The expansion and complexity of medical service have forced more consideration of the political aspects of medicine and the medical aspects of politics than ever before, and in the process have sensitized more physicians to political action, easing the transition into this formerly distasteful way of life. While the profession may have gained a degree of political sophistication, the marriage of medical philosophies and political practicalities is still a mixed one and subject to all the characteristic stresses thereof. Physician-politicians must not only enter a territory foreign to familiar professional functions but medical politics, while it may offer some indoctrination into the basics of political practice, does not afford as natural a transition into public politics as that found in other endeavors. But physician-politicians do provide medical expertise in the development of political action and, in so doing, learn to confront the same obstructions and exercise the same political maneuvers associated with nonmedical politics. They learn that politics in a white coat is still politics.

Whatever the relative state of the medical profession *vis à vis* the real world of politics, the profession has not reached the degree of political maturity where active recruitment of physicians to political awareness and action can be disregarded. One gets the impression that there is little depth to whatever progress has been made since we are admonished with regularity that physicians must involve themselves more. These calls are usually issued by those already involved and on the occasion of one or another of the political crises which seem to appear with frequency and emphasize to the active ones the dilatory natures of their colleagues. Again, the dissimilarity of medical practice and the full time political career appears. Only under unusual circumstances can the two be combined and one wonders, as the political demands propagate, when the physician ceases to exist and the politician takes over.

Contention is the substance of political activity and it is of a greatly different type than the physician is accustomed to. Whether as an office-seeker or in one of the many less visible roles, the politician must contend with others of differing but like ambition and, even when successful, maintain a continuing vigilance against others attempting to take over. Mandates, elective or otherwise, are rare and fragile at best. More often than not, political victories are determined by small numbers and can be destroyed the same way. And those who do achieve the state of grace face the continuing difficulty of reconciling

personal beliefs with those of the constituency, and it is often difficult to recognize the candidate in the office-holder.

Of all occupations, the practice of medicine is probably the least tolerant of sharing its practitioner with the political life. Politicians tend to incur the suspicions of their professional peers; the physician-politician who must represent a general constituency, who becomes indoctrinated into the realities of political achievement, and who becomes aware of the power of compromise as a political effector, may become thereby suspect in the eyes of the medical profession as having abdicated professional interests. Operating strictly from personal conscience (as the physician's background might direct) has a lofty connotation but can prove a lonely road and is often ineffective in this day when despite an occasional breach of the lines, party affiliation and fidelity remain among the most potent devices. Then one might say that medical politicians should function as public servants, sensitive to their constituents, opposed to special interests (which are always portrayed as inherently evil by their opponents — although everyone, even in medicine, is identified with one or another in some way). This too can dissipate the personal effort when it is not reconciled with practical solutions.

While the various social, business, and professional entities with their individual interests and functions make up the organic substance of politics, the whole relies for survival on its life blood — money. One could even define politics as the process of removing money from its source and returning it — less the handling charge — since it is difficult to think of any political activity that does not in the final analysis come down to a system for acquiring and dispensing money. Aspirants to public function (electoral or otherwise) may talk in terms of principles and worthy causes but the ultimate service rendered will be, directly or indirectly, the control of money — which is a potent attraction to the whole business. The form changes — pebbles, beads, pieces of bone, clay with cuneiform dents, metals of all kinds — but the significance of money and what it can do in the social and political world has always been paramount. Its use is the means of all political virtue and progress, and at the same time the source of political corruption and abuse. The net accomplishments of political action for the benefit of mankind can probably be measured by the extent to which the public, recognizing its vulnerability to it, exercises viable control over it.

Individuals or groups with special interests have always had a way of justifying the use of money to apply political pressure in favor of those interests. In our own time, the ease with which such pressures could become debasing has been recognized and, modest though they are, some efforts have been made to contain them. The procedure has been not only to prevent the easier forms of misuse of money but to keep under surveillance the legitimate methods by which groups collect and expend money in the interest of political effect, thereby limiting the possibilities of illicit use. Limitations on personal contributions have been established but more significant has been the prohibition of organizational funds (whose amounts could be hidden in various ways). This has resulted in the prompt appearance of other organizations, given the name of political action committees, whose purpose is to do the same thing but under close scrutiny and full recognition by the public of their source and purpose. Cynics may contend that the change is more in appearance than substance, but the maneuver has opened the process to financial accounting and publicity, probably as effective control as an imperfect society can devise.

Which brings us to our concern of the moment, which is not so much with those directly operating the political machinery as with the group that has evolved in the process of putting them there — the political action committees, our own in particular.

In the case of KaMPAC, the Board that runs it is worthy of a degree of gratitude on the part of the physicians of the state — although expressions of such gratitude in terms of financial support are far less than they should be (one similarity to the pre-PAC days and one reason for our prolonged preamble about money). For one thing, the members must be sustained by a degree of dedication to the system since they receive no remuneration other than the satisfaction of performing a valuable service. It is no easy task since the purpose is not simply distribution of financial support to candidates in the sometimes mindless form of party contributions. It entails the derivation of valid medical purposes (and neither denial nor apology is necessary that these conform to the interests of the Kansas Medical Society and consequently, Kansas physicians). It involves the evaluation of candidates in terms of their sympathy — or at least tolerance — to such purposes, the correlation of medical interests with other significant political issues, reconciliation with the political realities (including the inevitable pressures of party politics since the members represent both parties), and consideration of all candidates. KaMPAC represents one of the prime expressions of medical politics and, although the critics of medicine refuse to acknowledge it, the fundamental purpose of medical politics

is the furtherance of patient care, and the basic function of the medical political action committee is to translate this purpose from the medical level to general politics.

This is a formidable order and, like much public service, somewhat masochistic: the execution of these duties carries a high certainty of inviting disagreement from one quarter or another — with consequent refusal of some dissenters to participate. The dissenters deny themselves a respected political voice but rely perhaps on personal contributions of time and effort. While this is a legitimate approach, the gain in independence is lost in the posture.

1982 is an election year, and you may expect to hear from KaMPAC. The larger the group, the more certain it is to develop supportable medical objectives and produce a valuable medical effort. — *D.E.G.*

Abstracts

(Continued from page 26)

of neurologic examination were normal. Acid and alkaline phosphatase levels were elevated. Biopsy of the prostate showed adenocarcinoma. Results of indirect laryngoscopy and esophagogastroscopy were normal. Esophageal motility studies showed normal esophageal sphincters and weak peristaltic waves in the body of the esophagus. Electrodiagnostic studies showed that mean duration of motor unit potentials were shortened by 35 per cent. Repetitive supramaximal stimulation revealed the amplitude of initial evoked potential to be 0.8 mv on 30/sec stimulation. Facilitation occurred to greater than 300 per cent consistent with ELS. Biopsy of the mass in the clavicle showed adenocarcinoma, positive for acid phosphatase stain. With guanidine therapy dysphagia, electrodiagnostic and esophageal motility showed improvement. After bilateral orchiectomy and estrogen therapy, guanidine was discontinued. The patient remained asymptomatic.

Dysphagia was the only symptom of ELS in our patient. ELS should be considered in the differential diagnosis of dysphagia in a patient with any malignant disease. Another interesting feature of this case was the presence of bone metastasis which simulated primary bone tumor.

Recurrent Pleural Effusion in Congestive Heart Failure: Treatment by Pleurodesis

Frank X. O'Sullivan, M.D. and Marvin Dunn, M.D., Kansas City, Kansas.

Transudative pleural effusions are a common manifestation of such salt-retaining and hypervolemic states as cirrhosis, congestive heart failure, and nephrotic syndrome. Very little information regarding the direct treatment of these transudative pleural effusions can be found in the literature, probably due to the fact that most readily resolve with therapy directed at their underlying cause.

A 72-year-old white female developed clinical and radiologic signs of congestive heart failure in 1967. Subsequent cardiac catheterization revealed normal coronary vasculature and biventricular enlargement consistent with congestive cardiomyopathy. Conventional therapy controlled the patient's symptoms until three years prior to admission when a recurrent right-sided pleural effusion developed. The patient's condition subsequently deteriorated as her congestive heart failure and pleural effusion became refractory to optimal doses of digitalis, diuretics and vasodilators, necessitating increasingly more frequent admissions to the hospital. Finally, pleurodesis with tetracycline was performed in an attempt to relieve the patient's severe respiratory symptoms. This resulted in symptomatic, clinical, and radiologic improvement. Significant pleural effusion has not recurred. Pleurodesis should be considered a viable modality in the therapy of recurrent transudative pleural effusions.

The Preferential Use of Digitoxin in the Digitalization of Patients With Paroxysmal Atrial Fibrillation and Flutter

George Markus, M.D. and Marvin Dunn, M.D., Kansas City, Kansas

Digoxin controls the ventricular response in patients with atrial fibrillation and atrial flutter by slowing A-V nodal conduction. We have seen three patients with paroxysmal atrial flutter and fibrillation whose ventricular rate could not be controlled with digoxin. Up to one milligram per day, in divided doses, was used in all patients, with one patient receiving 3.5 mg/day. In two patients, therapeutic digoxin levels were not achieved (0.8 ng/dl-2.0 ng/dl). In the third patient, levels were not available. One patient had digoxin balance studies which did not demonstrate a problem with absorption or excretion. Subsequently each patient was digitalized with digitoxin, with a variable loading dose, and maintained on 0.1 mg/day. Each patient, in turn, manifested less frequent episodes of paroxysmal atrial fibrillation and a slower ventricular response during these episodes. Two achieved therapeutic blood levels of digitoxin. In the third, blood levels were not available. We conclude that patients with paroxysmal atrial fibrillation and flutter unaffected by digoxin may be well-controlled with digitoxin. The mechanism for this variation is unknown.

Percutaneous Transluminal Angioplasty in the Ischemic Limb

Joseph P. Galichia, M.D. and Roger W. Roberts, D.O., Wichita

Percutaneous transluminal angioplasty (PTA) was performed in 19 patients with lesions involving the iliac or superficial femoral vessels. The mean age of the patient population group was 63 years. Eleven patients had hypertension and 14 were heavy smokers. Attempted dilatations were performed on a total of 31 lesions. Dilatation of 17 iliac artery stenoses and 14 superficial femoral stenoses, of which four were totally obstructed, were attempted. Ninety per cent of attempted dilatations were successful resulting in significant symptomatic improvement in 84 per cent of patients. Clinical, hemodynamic, and angiographic improvement was obtained in 17 of 17 patients with high grade stenoses of the iliac arteries. PTA was successful in 11 of 14 superficial lesions. Of the three patients in whom dilatation could not be achieved, two superficial femoral arteries could not be cannulated. Only one complication was encountered in this series — a hematoma at the puncture site which was conservatively managed. It is concluded that PTA of iliac and femoral vessels can be carried out safely, quickly, and successfully in most patients.

Cardiobacterium Hominis Endocarditis Occurring on a Porcine Mitral Valve Prosthesis

Douglas K. Boehm, M.D. and Marvin Dunn, M.D., Kansas City, Kansas

Non-perioperative endocarditis occurring on porcine valves is

unusual and poorly described. *Cardiobacterium hominis* is also an unusual cause of endocarditis and has not been associated with any other disease. The patient presented had infectious endocarditis on a porcine mitral valve due to *C. hominis*.

A 52-year-old woman with a three-month history of lethargy, fever and chills, night sweats, and a 13.6 kg weight loss was admitted for suspected endocarditis. She had a history of rheumatic heart disease which necessitated tricuspid and mitral porcine valve prostheses in 1973. She had no history of recent dental manipulation or trauma. Peripheral petechiae, hepatosplenomegaly, elevated systemic and pulmonic venous pressures, and a new diastolic murmur were present. Echocardiography showed mitral valve vegetations. *Cardiobacterium hominis* was isolated from the blood and bone marrow. Heart failure developed due to acute mitral insufficiency despite antimicrobial therapy, and the tricuspid, mitral, and aortic valves were surgically replaced. At operation the mitral valve was found to be involved with endocarditis. The patient recovered after operation and penicillin therapy, and has resumed her usual activities.

Cardiac Surgery in Older Patients

M. Martin Halley, M.D.; Paul H. Kindling, M.D.; John W. Long, M.D. and Norman W. Thoms, M.D., Topeka

Open heart surgery has become more common in older age groups, due to the increasing age of the general population and technical achievements. Older patients and their families additionally desire surgical invertention when a reasonable chance of improvement in symptoms and prognosis exists.

This presentation looks at the results of our first 1,000 cases, comparing patients 64 years and younger with those 65 years and older as to types of procedures performed, operative mortality, and long term results in terms of mortality, morbidity, work, or retirement status.

Eight hundred eighty-eight coronary artery grafting procedures, 97 valve, and 15 other procedures were performed. Six hundred forty-eight (65%) were in the younger group and 352 (35%) were in the older group. The procedures evaluated were coronary artery bypass grafting, coronary artery bypass and ventricular aneurysm resection, reoperation for coronary artery bypass or valve procedure, valvular surgery, and miscellaneous procedures.

Operative mortality rate in uncomplicated coronary artery bypass grafting was 0.2 per cent in the younger group and 1.8 per cent in the older group for an overall mortality rate of 0.7 per cent. Ventricular aneurysm resection and reoperation showed no significant difference in mortality between age groups. The long term results in terms of return to work in the younger group and active retirement for the older age group appeared to be comparable. Actuarial analysis of long term survival for coronary artery disease has revealed a survival rate approaching that of age and sex matched populations. Long term survival of patients with valvular surgery showed significant improvement over non-surgically treated cases.

Encainide Therapy in Patients With Supraventricular Tachycardia

Robert L. Rinkenberger, M.D. and Marvin Dunn, M.D., Kansas City, Kansas.

The purpose of this study was to determine the electrophysiologic effects and efficacy of Encainide in patients with supraventricular tachycardia.

Two patients with recurrent supraventricular tachycardia refractory to conventional antiarrhythmic therapy had a control and repeat electrophysiology study to determine the electrophysiologic effects of Encainide. Patient number one had a nodoventricular accessory pathway, and patient number two had atrial fibrillation and atrioventricular reentry associated with manifest Wolff-Parkinson-White syndrome.

Encainide slowed AV nodal conduction and ablated supraventricular tachycardia in patient number one. The accessory pathway in patient number two was completely blocked in both the antegrade and retrograde direction, eliminating atrioventricular reentry and AP conduction during atrial fibrillation. Encainide has suppressed supraventricular tachycardia and has been well tolerated without side effects for six and eight months in patients number one and number two, respectively.

Conclusion: Encainide suppressed supraventricular tachycardia associated with an accessory pathway.

Suppression of Myelopoiesis in Legionnaire's Disease: A Case Report

Bruce Johnson, M.D.; Sean R. Lynch, M.D. and Chan Park, M.D., Ph.D., Kansas City, Kansas

A patient undergoing evaluation for moderately severe renal insufficiency developed fulminant five-lobed pneumonia due to *Legionella pneumophila*. Her clinical course was complicated by granulocytopenia. Bone marrow aspiration showed marked inhibition of myelopoiesis involving all stages of maturation. The presence of a serum inhibitor of granulopoiesis was demonstrated by in vitro bone marrow culture. Normal bone marrow cultured in the presence of serum from two normal controls (15% human serum ± 10% fetal calf serum) yielded 69 ± 5.2 (mean ± SE) and 61 ± 5 granulocyte colonies respectively. When the patient's serum was substituted for normal human serum only 14.3 ± 2.3 colonies were formed ($p < 0.001$). Each of the drugs to which she had been exposed was tested in the in vitro system using therapeutic concentrations and none showed significant suppression of granulocyte colony formation. These observations indicate that Legionnaire's disease was associated with a serum factor that markedly inhibited the growth of granulocyte colonies in this patient.

Intra- and Extra-Hepatic Biliary Ductal Obstruction and Combined Immunodeficiency in a Case of Acute Myelosclerosis

Ming-S. Lok, M.D.; Carol J. Trautt, M.T., A.S.C.P.; C. A. Chavez, M.D. and M. G. Rao, M.D., Leavenworth

A patient with acute myelosclerosis presented with pancytopenia, progressive jaundice, hypogammaglobinemia, and anergy. Following his death, autopsy showed the presence of fibrosis and myeloid metaplasia in the liver, spleen, and visceral lymph nodes. Numerous islands of myeloid metaplasia with occlusion of intrahepatic ducts, enlargement of periportal lymph nodes with compression of common duct, and cholestatic changes in the hepatic parenchyma were noted. These findings indicate that intra- and extra-hepatic biliary ductal obstruction was the cause of his cholestatic jaundice. The massive replacement of the lymphoid tissue by fibrotic and myeloid metaplastic process is probably the pathogenic mechanism of his lymphopenia and combined immunodeficiency.

The Detection of Anti-Platelet Antibodies by the ELISA

David Bodensteiner, M.D. and Fred Plapp, M.D., Kansas City, Kansas

The enzyme linked immunosorbent assay (ELISA) was used in the study of immune and drug induced thrombocytopenia (ITP). The technique of the platelet ELISA is simple; random donor platelets are incubated with the patient's serum, washed, incubated with an anti-IgG conjugated with alkaline phosphatase and followed by the addition of the substrate. The measured optical density is linearly proportional to the concentration of IgG per platelet. In six cases of clinical ITP, five had a positive platelet ELISA. Two cases of severe drug induced thrombocy-

topenia (platelet counts less than 20,000) were also studied. The ELISA initially was negative in one patient and slightly positive in the second. The patients were receiving quinidine and tobramycin/cephalexin, respectively. When the ELISA was repeated using the first patient's serum incubated with quinidine at therapeutic levels, it became strongly positive. In the second patient, there was no change with tobramycin but with cephalexin, the ELISA again turned strongly positive. Both patients now have normal platelet counts after discontinuing the drugs. We have found the platelet ELISA of value in diagnosing immune mediated and drug induced thrombocytopenia, and in determining the causative agent in the drug induced cases.

Life-Threatening Hemorrhage Due to Acquired Factor VIII Inhibitors in Non-Hemophiliacs: Report of a Case and Review of Current Therapies

Donna E. Sweet, M.D. and Harry E. Hynes, M.D., Wichita

Acquired inhibitors to Factor VIII may develop in previously healthy elderly persons, as well as in post-partum women and in those suffering from autoimmune diseases. This condition should be considered as a possible cause of any hemorrhagic condition in a patient with an isolated prolongation of the partial thromboplastin time (PTT) and no previous bleeding diathesis.

Our case is that of a 68-year-old male without previous bleeding disorder who presented with severe hemorrhage into both neck and retroperitoneum, and a prolonged PTT. Studies showed a Factor VIII level of 0 per cent and an excess of Factor VIII inhibitor. Results of all other coagulation studies were normal and there was no evidence of any autoimmune condition. Infusion of large amounts of Factor VIII did not improve his condition. Two-volume exchange plasmapheresis on multiple occasions led to only transient increases in Factor VIII levels. Addition of cyclophosphamide (2 mg/kg/d) and prednisone (60 mg/d) led to a gradual rise in Factor VIII level to 55 per cent, a normal PTT, and cessation of hemorrhage. Cyclophosphamide and prednisone were tapered and stopped during a three month period. One month thereafter, the Factor VIII level was 127 per cent and there were only trace amounts of inhibitor detectable. He has since remained free of bleeding problems.

Literature review supports our conclusion that successful treatment of hemorrhage due to Factor VIII inhibitors requires aggressive immunosuppression. Cyclophosphamide has proven to be more successful than either azothioprine or 6-mercaptopurine, and steroids, although shown to be helpful in some studies, were of questionable value in others.

Hemoglobinemia: A Contributing Factor to Elevated MCHC

Carol J. Trautt, M.T., A.S.C.P. and Ming-S. Lok, M.D., Leavenworth

Two patients with elevated mean corpuscular hemoglobin concentration (MCHC) were studied. Both patients were taking alpha-methyldopa and were found to have positive Coomb's tests. The first patient had active autoimmune hemolysis and approximately 20 per cent spherocytes in his peripheral blood. The second patient, who was subsequently diagnosed to have pernicious anemia, had significant intramedullary hemolysis and 1 per cent spherocytes. Although spherocytosis is a known cause of elevated MCHC, it is possible that hemoglobinemia as a result of hemolysis may also contribute to the elevation of the MCHC in these two patients. To study the effect of hemoglobinemia on MCHC, two in vitro models were designated. One model is equivalent to intravascular hemolysis, and the other depicts extravascular hemolysis. From the data provided by the in vitro models and these two patients it was concluded that hemoglobinemia, caused by either intravascular or extravascular hemolysis, would spuriously elevate the MCHC.

Case Report: Geomelophagia. An Unusual Pica in Iron Deficiency Anemia

Bruce E. Johnson, M.D. and Ronald L. Stephens, M.D., Kansas City, Kansas

A patient with lung cancer treated by radiation and in remission presented with a two-month history of compulsive eating of raw, chilled potatoes. Suspicion of a pica due to iron deficiency anemia was confirmed after complete laboratory evaluation. The source of iron loss was found to be gastrointestinal bleeding. Therapy with iron sulfate was started with a subsequent rise in the hemoglobin; the pica ceased within one week of initiation of therapy. If searched for, pica is a common manifestation of iron deficiency; however, this patient apparently represents the first report of geomelophagia. Appropriate investigation of compulsive eating habits might lead to the diagnosis of iron deficiency and also allay patients anxieties toward their behavior.

Correction of Leucocyte Dysfunction by Bethanechol Giving Successful Treatment of Hidradenitis Suppurativa

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Hidradenitis suppurativa (HS) infects the apocrine sweat glands giving chronic recurrent abscesses of axillary and perineal areas. We report a patient who had a defect in leucocyte (PMN) killing of bacteria leading to persistent HS. This PMN defect was corrected with a cholinergic agonist *in vitro,* and treatment with urecholine resulted in healing.

A 70-year-old white male was admitted with intractable HS of both axillae and perineum. Multiple previous courses of antimicrobials had had little effect. Examination of axillae and perineum revealed multiple abscesses, draining sinus tracts, and scars of previous drainage procedures. *E. coli,* enterococcus, proteus, staphylococcus, and bacteroides were cultured from the abscesses. Abscess drainage and sequential therapy with gentamicin (30 days), ceforanide (19 days), moxalactam (16 days), and cephalexin (5-6 weeks) had little success. Immunologic studies showed normal function of T and B cells, normal Ig and C levels, phagocytosis, and chemotaxis. PMN killing of staphylococci and *E. coli* was reduced (139 vs 3 CFU), but corrected with 10-6M carbachol, a cholinergic agonist. Treatment with oral bethanechol alone, 50 mg three times/day, without antibiotics for six weeks gave definite healing of the abscesses, ulcers, and fistulae. The patient's PMN killing was normal when tested at two, four, and six months during bethanechol therapy.

The mechanism in this patient was bethanechol-induced increase in cyclic-GMP impaired lysosomal function and thus reduced bactericidal killing. Cholinergic agonists corrected the *in vitro* abnormalities and were useful clinically in this patient.

A Case of Fatal Toxic Shock Syndrome

William F. Cathcart-Rake, M.D. and Robert W. Brown, M.D., Salina

A 27-year-old white female presented with a history of high fever, myalgias, malaise, nausea, vomiting, and diarrhea coinciding with her menstrual period. She had previously been healthy except for a similar illness coinciding with menstruation one month earlier. Upon presentation she was febrile, oliguric, appeared dehydrated, had erythema of her oropharynx, and had mild abdominal tenderness; no rash or adenopathy were found. She became hypotensive shortly after admission to the hospital. Peripheral smear showed a leukocytosis; sedimentation rate was markedly elevated; BUN, 69; and creatinine, 5.0. Multiple cultures were obtained and subsequently proved to be negative. After initial improvement in hypotension and oliguria with fluid therapy, she developed non-cardiogenic respiratory failure (ARDS), acidosis, and refractory hypotension. Despite intravenous fluids, correction of acidosis, broad spectrum antibi-

otics, pressor agents, pharmacological doses of corticosteroids, and mechanical ventilation with positive end-expiratory pressure, her condition deteriorated and she died 96 hours after hospitalization. Postmortem examination and review of all laboratory and cultural data failed to reveal the cause of her illness. The complex of symptoms and clinical findings are consistent with toxic shock syndrome.

Toxic Shock Syndrome With Severe Multisystem Involvement

David Webb, M.D.; J. D. Peterie, M.D.; Ray Hershberger, M.D.; D. L. Ross, M.D. and H. C. Goodpasture, M.D., Wichita

Toxic shock syndrome is a relatively new disease entity of significant clinical importance. It is associated with menses and also with colonization of the vagina with *Staphylococcus aureus* usually of phage group I or III. It has been defined as: (1) An abrupt onset febrile illness associated with vomiting and watery diarrhea; (2) Hypotension with a fall in systolic blood pressure to less than 90 mmHg; (3) Diffuse macular erythroderma followed by desquamation of the skin of the palms and soles in the second week; (4) Multiple involvement of three or more organ systems; and (5) Exclusion, both clinically and biochemically, of other common febrile illnesses.

We report an unusually severe case presenting with the above syndrome which progressed to vascular collapse and evidenced several unusual features including peripheral gangrene of upper and lower extremities, severe disseminated intravascular coagulation (DIC), and anuric renal failure of four weeks duration. During the acute phase of the illness, the patient's severe toxic state with refractory hypotension and adult respiratory distress syndrome appeared to respond to plasmapheresis, a treatment modality not previously utilized for this syndrome. The basic pathophysiology of this syndrome is unclear, but observations appear to suggest a circulating exotoxin which may precipitate the DIC, vascular collapse, and end organ dysfunction. Plasmapheresis may be of value in the management of severe cases of toxic shock syndrome.

Gram-Negative Bacteremias: Analysis of Factors for Clinical Assessment of Gentamicin Resistance

J. M. Lynch, M.D.; G. R. Hodges, M.D.; G. M. Clark, Ph.D. and D. L. Dworzack, M.D., Kansas City, Kansas

Charts of 163 patients with 183 episodes of gram-negative bacillary bacteremia were reviewed to determine a profile that would select patients at high risk for developing gentamicin-resistant gram-negative bacillary bacteremia. Gentamicin-resistant gram-negative bacilli were associated only with institution-acquired bacteremias, not community-acquired bacteremias. Among institution-acquired episodes, urinary tract infection (p less than 0.001), diagnostic or therapeutic procedures of the lower respiratory tract ($p = 0.036$) or urinary tract ($p = 0.024$), presence of pneumonic infiltrate on chest roentgenogram ($p = 0.029$), prior therapy with gentamicin (p less than 0.002), and prior therapy with other antibiotics (p less than 0.001) were significant risk factors. Because only two of 29 gentamicin-resistant bacteria were resistant to amikacin, we advocate initial treatment with amikacin for patients with evidence of institution-acquired gram-negative bacteremic episodes in our hospital. Gentamicin is still our initial choice for a community-acquired episode. While our findings may not be applicable to hospitals with low incidence of gentamicin-resistant bacteremia, we show that epidemiologic data from an infection control program can be utilized to develop therapeutic strategy.

Atypical Measles (Rubella) Syndrome in an Adolescent

William R. Alsop, M.D.; H. C. Goodpasture, M.D. and T. W. Smith, M.D., Wichita

A syndrome of atypical measles (rubeola) has been described for a number of years in children and more recently in adolescents and young adults, especially those who received the killed measles vaccine marketed prior to 1967. These patients invariably show a dramatic antibody response to rubeola antigens.

We report a 17-year-old white male who developed an acute illness characterized by fever, peripherally accentuated erythematous rash, inflammation of the mucous membranes, jaundice with evidence of hepatocellular necrosis and acute non-cardiac pulmonary edema, who showed no evidence of antibody response to rubeola (complement fixation — acute 1:16, convalescent 1:8), but a dramatic titer rise to the rubella virus, from 1:40 to 1:320 in one week's time. The patient had received the live attenuated rubella virus vaccine nine years earlier. Infections due to cytomegalovirus, herpes simplex, toxoplasmosis, Rocky Mountain spotted fever, and Q fever were ruled out on the basis of negative serologic studies. This illness responded dramatically to corticosteroids, and the patient recovered without sequelae.

This is the first report of atypical rubella. Our paper will present data documenting the illness in this patient and will compare and contrast this syndrome to the atypical rubeola syndrome.

Alternaria Osteomyelitis of the Hard Palate and Maxillary Sinus: Report of a Case and a Review of the Literature

B. Ellis, M.D., H. C. Goodpasture, M.D.; G. Randall, M.D. and T. Carlson, M.D., Wichita

Alternaria is a fungus that is ubiquitous in the air and soil and is often isolated from normal skin. It is generally regarded as non-pathogenic for humans, but can cause allergic reactions such as asthma or hypersensitivity pneumonitis. We report maxillary osteomyelitis due to alternaria with contiguous involvement of the hard palate in a healthy 31-year-old white male. The diagnosis was confirmed by biopsy and histologic studies showing typical organisms within granulomas and recovery of the organism from the cultured biopsy. Study of this patient's lymphocytes against a standard panel of antigens showed a normal response except for alternaria where there was only a minimal response. This *in-vitro* finding was paralleled by study of the biopsy which showed a minimal infiltrate with lymphocytes. Miconazole given intravenously for one month failed to cure this infection, but the organism was eliminated with healing of the lesion following six weeks of intravenous amphotericin. In addition the *in-vitro* mitogen response to alternaria improved during this time as did the histologic response to the organism as measured by a lymphocytic infiltration of the involved tissue.

This paper will review the findings in this patient in detail as well as the literature regarding alternaria infections, with particular focus on the other reported case of maxillary sinus osteomyelitis.

Relapsing Polychondritis With Glomerulonephritis

James L. Ruhlen, M.D.; Kent A. Huston, M.D. and William G. Wood, M.D., Kansas City, Missouri

A 32-year-old man presented with relapsing polychondritis (RP) and microhematuria. Renal biopsy disclosed focal segmental glomerulonephritis with occasional crescents. Immunofluorescent and electron micrographic studies suggested immune complex mediated glomerular injury. Initially, life-threatening upper airway obstruction responded to high-dose corticosteroids. Subsequently, progressive renal insufficiency

and proteinuria due to increasingly active glomerulonephritis were treated by the addition of cyclophosphamide, with sustained improvement. This case supports the concept that RP is an immunologically mediated disease, and suggests that a regimen of prednisone and cyclophosphamide warrants consideration for use in cases of RP with glomerulonephritis.

Plasmapheresis in the Treatment of Cryoglobulinemia

Patrick Wolf, M.D.; Dennis L. Ross, M.D. and Kenneth W. Bruner, M.D., Wichita

Cryoglobulins are found in a wide variety of clinical diseases, usually reflecting immunologic disorders. Symptoms attributed to cryoglobulins are diverse including vasomotor, cutaneous, renal, neurologic, thrombotic, and hemorrhagic phenomena.

A 63-year-old female presented with symptoms of peripheral sensory neuropathy and petechiae. She was found to have cryoglobulinemia and cryofibrinogenemia associated with Sjögren's syndrome. Due to progression of symptoms despite aspirin, steroids and immunosuppressives, she received plasmapheresis with transient (2-3 weeks) resolution of symptoms.

Pre and post plasmapheresis immunologic profiling and nerve conduction studies were obtained. Treatments were done four consecutive days initially, and subsequently done monthly, using albumin and fresh frozen plasma as replacement solutions for removed plasma.

Characterization of the cryoglobulin included immunoelectrophoresis showing monoclonal IgM and polyclonal IgG with the cryoprecipitate showing strong rheumatoid factor activity. Complement studies suggested evidence of classical pathway activation. Concomitant with improvement of symptoms, tests for serum cryoglobulinemia became negative.

Clinical uses of plasmapheresis are currently being defined. Plasmapheresis appears to be a very effective and benign adjunctive treatment in the management of symptomatic cryoglobulinemia.

Systemic Manifestations of Pseudogout

Suzanne Wedel, M.D. and Marvin Dunn, M.D., Kansas City, Kansas

Pseudogout has been labeled "the great immitator." It has five clinical presentations: (1) Type A — intermittent attacks of acute arthritis with asymptomatic intervals between attacks; (2) Type B — continued acute attacks involving several joints; (3) Type C — progressive chronic arthritis with superimposed acute attacks; (4) Type D — chronic arthritis in the absence of acute attacks; and (5) Type E — asymptomatic patients with condrocalcinosis and calcium pyrophosphate dihydrate crystals (CPPD) in the synovial aspirate.

A 76-year-old man presented with fever and a right lower lobe infiltrate on chest x-ray. He was treated with a two-week course of antibiotics and chest x-ray showed improvement, but he continued to have daily spiking fevers. Blood, CSF, urine, sputum, and bone marrow cultures were negative. The sedimentation rate was 113 mm/hr. He had swollen erythematous joints with significantly decreased range of motion. Synovial fluid analysis revealed 44,000 WBC/−80% polymorphonuclear leukocytes. Cultures were negative. CPPD crystals were present. X-rays showed degenerative joint disease and chondrocalcinosis. The patient was treated with intravenous colchicine and indomethocin. Subsequently he became afebrile and joint mobility returned to normal.

Reactive Polyarthritis Due to *Yersinia Enterocolitica*

Larry Rumans, M.D.; Marla Newell, M.T., A.S.C.P.; Elbie Loeb, M.D.; Raymond Lumb, M.D. and Robert Jacoby, M.D., Topeka

A wide variety of clinical manifestations of disease may be attributed to infection with *Yersinia enterocolitica* including acute septicemic and subacute localizing types. Reactive rheumatic polyarthritis may occur in some individuals, and the clinical picture of Yersinia arthritis may be modified by the genetic background associated with the histiocompatibility antigen B27 (HLA-B27).

We report a patient who developed an overt Reiter's syndrome including signs of ocular inflammation and nonsuppurative polyarthritis accompanied by fever in whom the triggering event appeared to be a clinically insignificant acute diarrheal illness of short duration. Routine culture techniques were nonrewarding; however, *Yersinia enterocolitica* was recovered when stools were incubated utilizing cold enrichment techniques and weekly subculture to appropriate media performed. Recovery followed specific therapy.

B27-positive individuals appear to react in an unusual way to a variety of infective agents. In addition, the frequent onset of Yersinia arthritis in patients with Reiter's syndrome and ankylosing spondylitis and the knowledge that several rheumatic diseases with B27 positivity are associated with some infections lend support to the precipitating role of certain infections — *e.g., Yersinia* — to the development of spondylytic processes. Even within this subgroup of individuals, however, there are certain clinical characteristics of the rheumatic process which may focus attention on the infectious nature of the triggering event. Such illnesses may be more common than are ordinarily appreciated.

Primary Hypothyroidism: Suprasellar Pituitary Enlargement and Regression on CT Scanning

B. J. Silver, M.D.; J. L. Kyner, M.D.; A. R. Dick, M.D. and C. H. J. Chang, M.D., Ph.D., Kansas City, Kansas

Pituitary enlargement and hypertrophy is known to occur secondary to chronic, endocrine target organ failure (*i.e.* hypothyroidism, Nelson's syndrome), but such cases have all been diagnosed from radiological enlargement of the bony sella. We believe this is the first reported case of pituitary enlargement due to 1 degree hypothyroidism without concomitant changes in the bony sella.

A 23-year-old woman, felt to be adequately thyroxine-replaced, presented with unrelated optic neuritis. Her hypothyroidism had resulted from a thyroidectomy done ten years previously for "goiter." Cranial CT scan, to evaluate her neurologic process, showed suprasellar pituitary enlargement, although tomograms of the sella turcica were normal. She had few clinical symptoms or signs of thyroid deficiency (which is typical of many young hypothyroid patients), but was found to have 1 degree hypothyroidism, with a serum T4 of 0.4mg/dl and a TSH of 848 μU/ml (normal 0-6). Thyroxine replacement was closely supervised, and two months later a repeat CT scan showed that pituitary size was normal; pituitary endocrine evaluation at this time was also completely normal. Serum T4 was 10.8 mg/dl and TSH was 0.1 μU/ml, evidence for physiologic replacement.

With increasing availability of CT scanning, early or subtle changes in the pituitary may be found before there are bony sella changes, and the possibility of abnormality due to primary endocrine organ failure should be considered. This is an example of a pituitary "tumor" that can be treated medically, rather than with surgical intervention.

Non-Anion Gap Metabolic Acidosis Complicating Recovery From Diabetic Ketoacidosis

Jon Klein, M.D.; Barbara P. Lukert, M.D.; Joseph L. Kyner, M.D. and R. Neil Schimke, M.D., Kansas City, Kansas

Two patients on eight occasions developed a hyperchloremic non-anion gap metabolic acidosis during therapy for diabetic ketoacidosis. On each occasion the clinical presentation was

consistent with a distal (Type I) renal tubular acidosis (RTA). That disorder is characterized by an inability to acidify the urine. As in Type I RTA, these patients exhibited an elevated urinary pH with an associated hyperchloremic non-anion gap acidosis. While the mechanism is unclear, all patients were profoundly hypophosphatemic, a state reported to cause renal tubular dysfunction.

This represents an infrequently recognized condition with appropriate therapy different from that required for diabetic ketoacidosis. Administration of normal saline and sodium bicarbonate in these patients resulted in transient hypernatremia without correction of the acidosis. Proper therapy would seem to be directed at correction of hypophosphatemia and administration of bicarbonate precursors lacking in sodium.

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Hypergastrinemia and ZES

(Continued from page 20)

Answers

1. See *Table I*.
2. a. Elevated fasting gastrin
 b. Gastric acid hypersecretion
 c. Positive secretin stimulation test
3. See *Table III*.
4. 6 hour, hourly gastric analysis following administration of cimetidine +/− anticholinergic at doses being used
5. Any of the following, especially occurring after vagotomy or other prior gastric surgery:
 a. Failure to suppress ZES symptoms or complications on adequate medical regimen
 b. Patient refuses life-long medical regimen
 c. Patient non-compliant with medical regimen
 d. Unacceptable drug side effects on medical regimen

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INDICATIONS: *Therapeutically* (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: • infected burns, skin grafts, surgical incisions, otitis externa • primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia) • secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis) • traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically,* the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: Not for use in the eyes or in the external ear canal if the eardrum is perforated. This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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Motrin® vs aspirin w/codeine...
(ibuprofen)

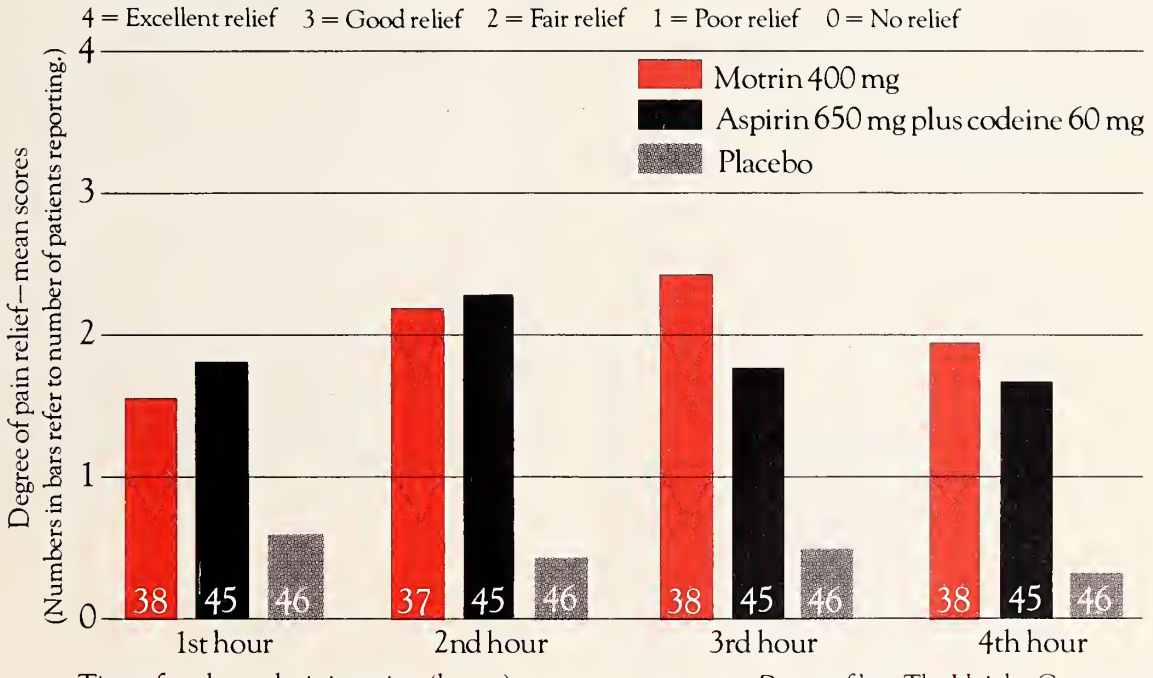


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compare the analgesic effect

A *Motrin* 400 mg dose relieved postsurgical dental pain as effectively as a combination of 650 mg aspirin and 60 mg codeine (two aspirin-with-codeine No. 3 tablets) in a study of 129 patients. In this double-blind, placebo-controlled, randomized study, no statistically significant difference in relief of pain was noted at 1, 2, and 4 hours between the *Motrin* and aspirin-with-codeine groups... with *Motrin* being significantly more effective ($p = 0.03$) at the three-hour interval. Active treatment was significantly more effective ($p < 0.0001$) than placebo at all time intervals.

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Please turn the page for a brief summary of prescribing information.

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Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Relief of mild to moderate pain.

Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin:* Used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

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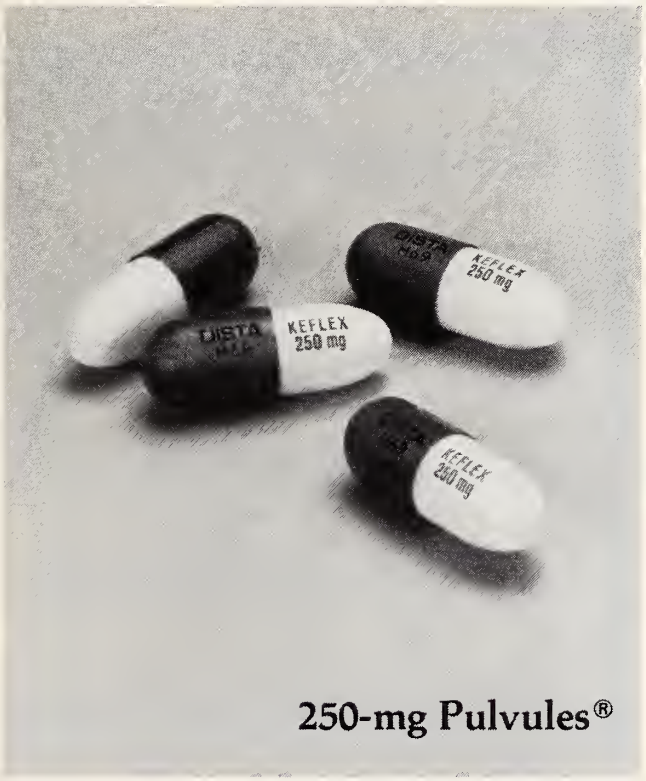
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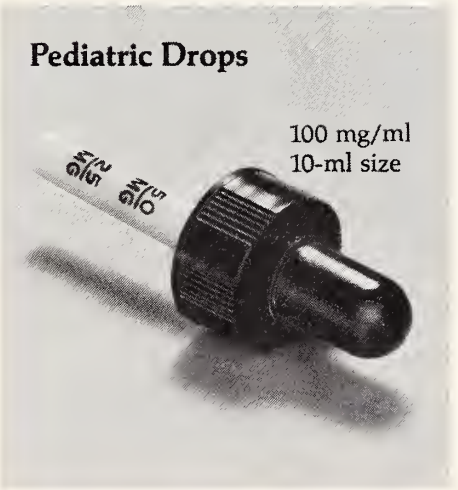
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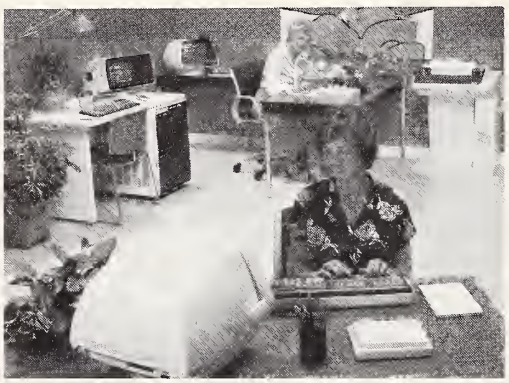
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Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. Limited clinical information presently available on effectiveness of treatment of otitis media with Bactrim when infection is due to ampicillin-resistant *Haemophilus influenzae*. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

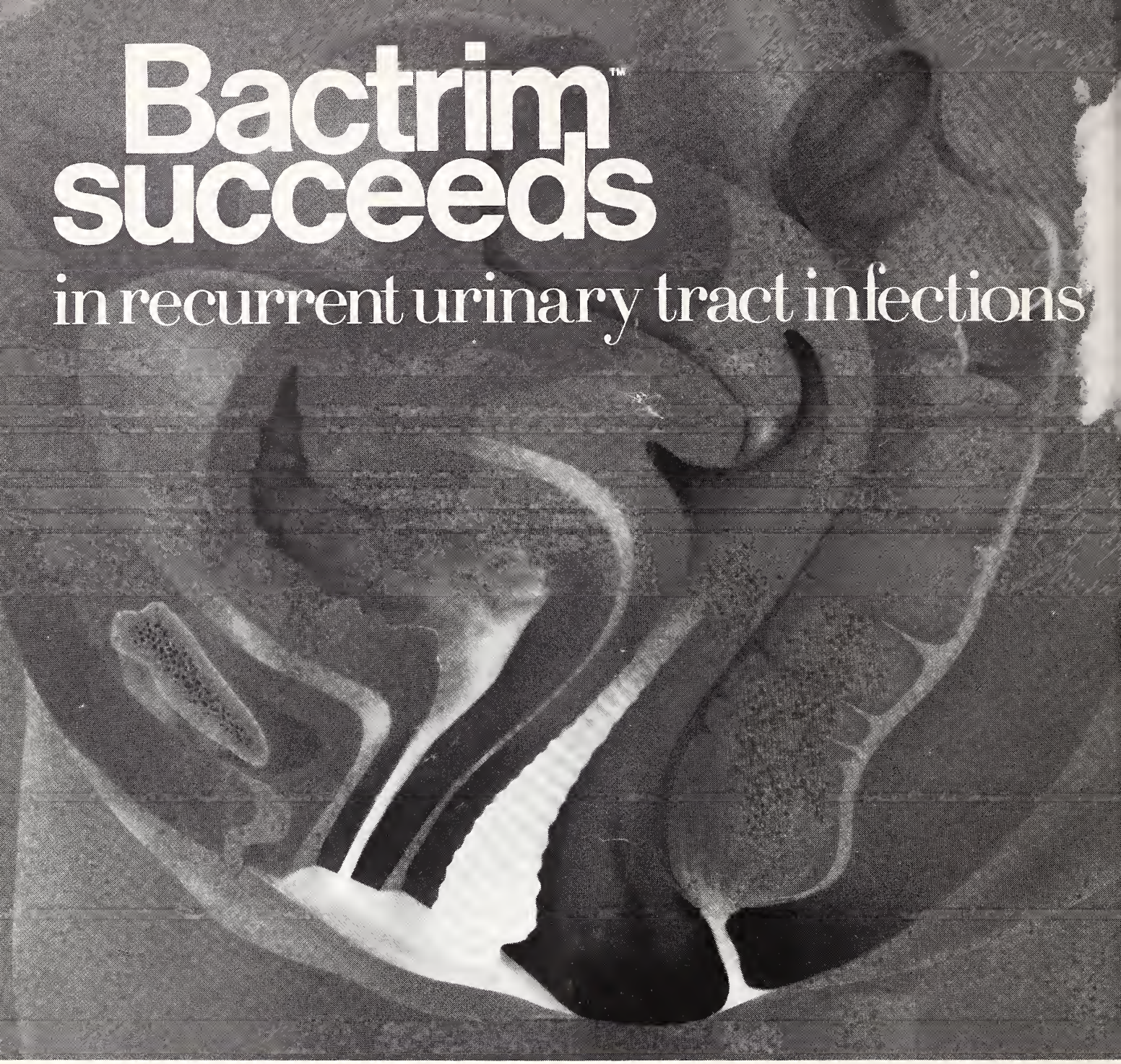
Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets*, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets*, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry-flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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Bactrim™ succeeds

in recurrent urinary tract infections*

from site to source

Bactrim continues to demonstrate high clinical effectiveness in recurrent urinary tract infections. Bactrim reaches effective levels in urine, serum, and renal tissue[1]...the trimethoprim component diffuses into vaginal secretions in bactericidal concentrations[1]... and in the fecal flora, Bactrim effectively suppresses Enterobacteriaceae[1,2] with little resulting emergence of resistant organisms.

1. Rubin RH, Swartz MN: *N Engl J Med 303*:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

Bactrim™ DS

160 mg trimethoprim and 800 mg sulfamethoxazole

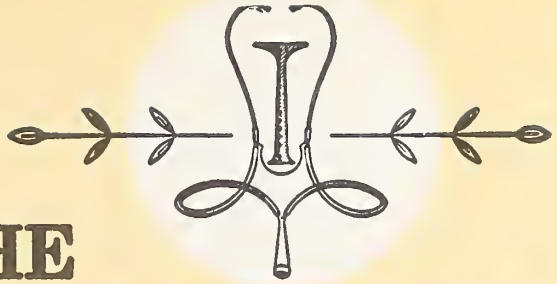
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maximizes results with B.I.D. convenience

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* due to susceptible strains of indicated organisms

Please see previous page for summary of product information.



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AMA House of Delegates

Summary of Actions Taken at the 1981 Interim Meeting, Las Vegas

The 1981 Interim Meeting of the American Medical Association was held on December 6-9 at the Las Vegas Hilton Hotel. The Kansas Medical Society was officially represented by its full complement of delegates (the undersigned), as well as Herman W. Hiesterman, M.D., President, and Jerry Slaughter, Staff. Also present from Kansas was William J. Reals, M.D., Wichita, representing the College of American Pathologists.

The 282 Delegates present represented state medical organizations as well as 61 specialty societies. Six new national specialty societies were voted into representation at this meeting. They are:

American Association of Clinical Urologists
American College of Nuclear Physicians
American Orthopaedic Association
American Society of Gastrointestinal Endoscopy
American Society of Therapeutic Radiologists
Association of Life Insurance Medical Directors of America

Kansas was successful in promoting the candidacy of William J. Reals, M.D., Wichita, for a seat on the AMA Council of Medical Education to fill an unexpired term. This council is concerned with undergraduate, graduate, and postgraduate education in the United States. It is comprised of 12 physicians and staff members and meets regularly to discuss medical education matters and to advise the AMA on major policy issues. We believe Dr. Reals is the first Kansas Physician to be elected to a major AMA position.

Some of the major issues considered by the House were the following:

Scientific Reports

The AMA Council on Scientific Affairs submitted several reports on issues of general interest to physicians and the public. One report calls for improved care for homosexuals. It concludes that nonjudgmental recognition of sexual orientation enhances the physician's ability to render optimal patient care in health as well as in illness. Other scientific reports concerned the establishment of guidelines for estrogen replacement in menopausal women; causative factors contributing to depression especially among women; and progress in DMSO research.

Health Care Coalitions

The House endorsed a statement by six national organizations encouraging the development of voluntary health care coalitions. The statement urges local coalitions to analyze resources and establish projects to seek solutions to health care problems. It is believed that this action will have significant implications for the medical profession. The coalitions represent a continuation of the AMA corporate liaison activity begun three years ago. Besides the AMA, other participating organizations are the American Hospital Association, Blue Cross-Blue Shield, Business Roundtable, Health Insurance Association of America, and AFL-CIO.

Competition Legislation

The House received two comprehensive reports on the Congressional and Administration activities regarding "pro-competition/consumer choice" proposals. In adopting the reports, the Delegates renewed AMA's strong concern that such legislation must be monitored carefully. The AMA objective is to preserve accessibility of high quality health care delivery. The House directed the AMA Board and Councils to act to assure the strengths of the existing system of free enterprise medicine.

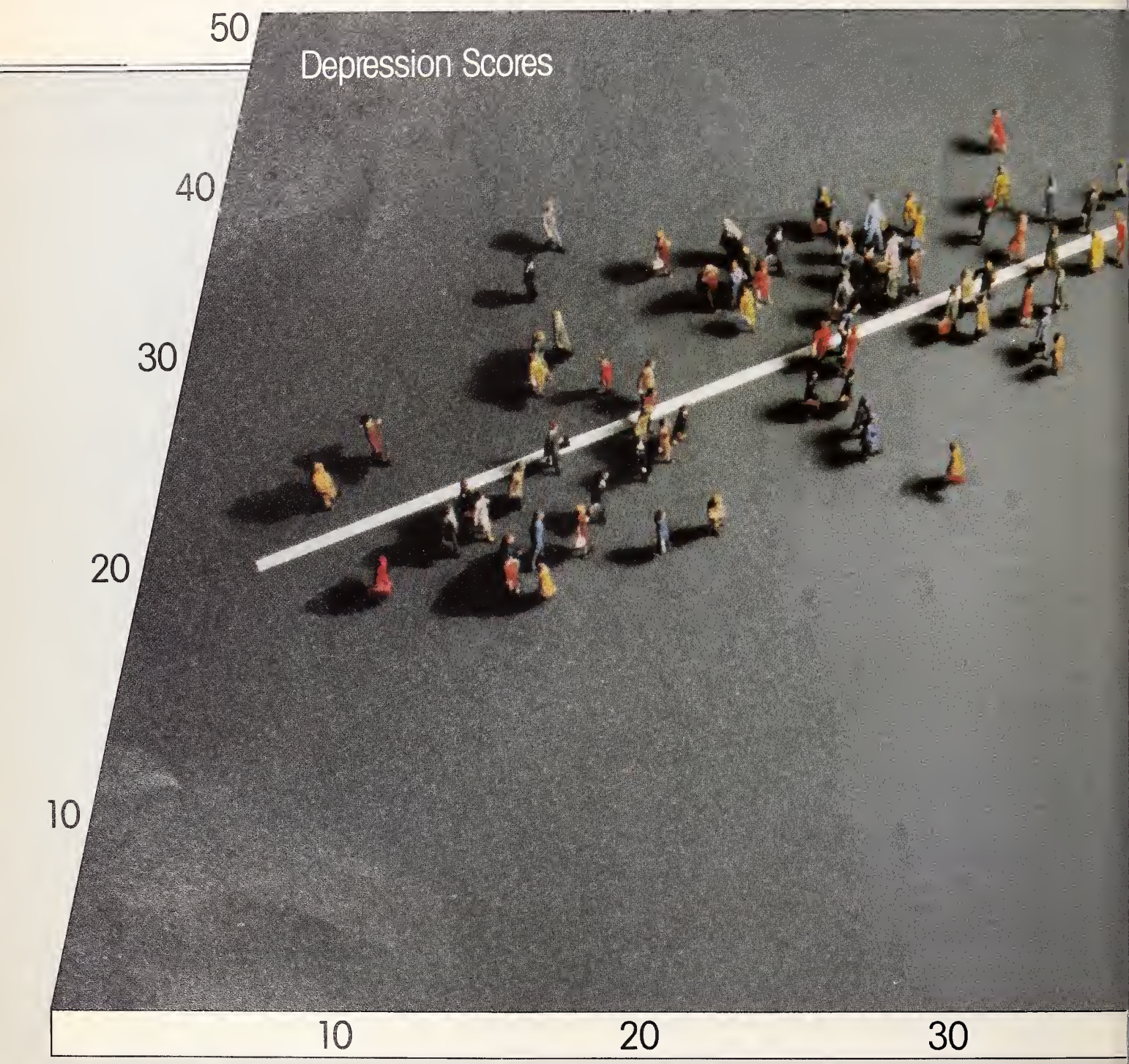
Voluntary Peer Review

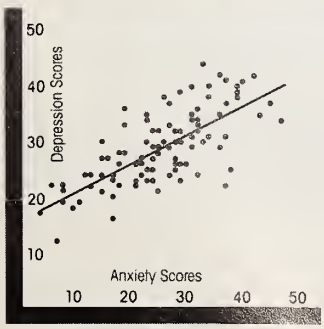
We reaffirmed medicine's continuing commitment to the development and maintenance of voluntary, professionally-directed peer review. In a related action, the House adopted a set of principles for voluntary peer review. These principles should be found in any peer review organization or system. The objective is to assure the quality and appropriateness of medical care services. The principles are as follows:

1. Medical peer review is an organized effort to evaluate and analyze medical care services delivered to patients and to assure the quality and appropriateness of these services. Peer review exists to maintain and improve the quality of medical care.
2. Medical peer review is a local process.
3. Physicians are ultimately responsible for all peer review of medical care.
4. Physicians involved in peer review should be

(Continued on page 85)

FOR THE 7 OF 10 NONPSYCHOTIC





Clear correlation between anxiety and depression[3]

The above graph illustrates a relationship between anxiety and depression, indicating that patients seldom present with anxiety or depression alone; more often they have both in varying degrees. Data based on a sampling of 100 outpatients (64 male; 36 female) seen at a general psychiatric clinic.

[3]Adapted from Clagharn, J. The anxiety-depression syndrome. *Psychosomatics 11*:438-441, Sept-Oct 1970.

ROCHE

DEPRESSED PATIENTS WHO ARE ALSO ANXIOUS[1,2]

Most depressed patients are also anxious...

Some authors estimate that 70% of all nonpsychotic patients with symptoms of depression have concomitant symptoms of anxiety.[1,2] One author found a distinct correlation between anxiety and depression scores in 100 nonpsychotic outpatients administered the Minnesota Multiphasic Personality Inventory in a general psychiatric clinic.[3] As depression scores increased, so did anxiety scores. No attempt was made to select patients other than to exclude psychotics.

but not psychotic

The logic of treating both components of anxious depression is clear. Antipsychotics, like the phenothiazines, however, carry a well-documented risk of tardive dyskinesia.[4] Because of this, an APA Task Force recently recommended the judicious use of phenothiazines in cases other than chronic psychosis or the use of alternative treatments.

A better way to give relief

Limbitrol combines the specific anxiolytic action of Librium® (chlordiazepoxide HCl/Roche)—a benzodiazepine with a long history of safe use—with the antidepressant action of amitriptyline, a tricyclic of established clinical efficacy. In comparison to phenothiazines, Limbitrol and its components have rarely been associated with tardive dyskinesia or other extrapyramidal side effects. And in terms of rapid response and patient compliance, Limbitrol appears to be superior to amitriptyline alone. Controlled multiclinic studies showed Limbitrol relieved more symptoms more rapidly than did amitriptyline.[5] Despite a higher incidence of drowsiness, the dropout rate due to side effects was lower with Limbitrol. (See adverse reactions section in summary of product information on next page. As with any CNS-acting agent, patients should be cautioned about driving or using dangerous machines while on therapy with Limbitrol.)

References: 1. Rickels K. Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine,* ed. Jarvik ME. New York, Appleton-Century-Crofts, 1977, p. 316. **2.** Schatzberg AF, Cole JO: Benzodiazepines in depressive disorders. *Arch Gen Psychiatry 35:*1359-1365, 1978. **3.** Claghorn J: The anxiety-depression syndrome. *Psychosomatics 11:*438-441, 1970. **4.** The Task Force on Late Neurological Effects of Antipsychotic Drugs: Tardive dyskinesia, summary of a task force report of the American Psychiatric Association. *Am J Psychiatry 137:*1163-1172, 1980. **5.** Feighner JP *et al.* A placebo-controlled multicenter trial of Limbitrol versus its components (amitriptyline and chlordiazepoxide) in the symptomatic treatment of depressive illness. *Psychopharmacology 61:*217-225, 1979.

Anxiety Scores

50

In moderate depression and anxiety

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Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Relief without a phenothiazine

Please see summary of product information on next page.

LIMBITROL® TABLETS Tranquilizer–Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use, then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated. sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion at daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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Tablets

DESCRIPTION

Each prolonged action tablet contains:

Phenylephrine Hydrochloride	25 mg
Phenylpropanolamine Hydrochloride	50 mg
Chlorpheniramine Maleate	8 mg
Hyoscyamine Sulfate	0.19 mg
Atropine Sulfate	0.04 mg
Scopolamine Hydrobromide	0.01 mg

Ru-Tuss Tablets act continuously for 10 to 12 hours.

Ru-Tuss Tablets are an oral antihistaminic, nasal decongestant and anti-secretory preparation.

INDICATIONS AND USAGE Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues. Phenylephrine and phenylpropanolamine combine to exert a vasoconstrictive and decongestive action while chlorpheniramine maleate decreases the symptoms of watering eyes, post nasal drip and sneezing which may be associated with an allergic-like response. The belladonna alkaloids, hyoscyamine, atropine and scopolamine further augment the anti-secretory activity of Ru-Tuss Tablets.

CONTRAINDICATIONS Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.

WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

PRECAUTIONS Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with glaucoma, or urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See Warnings).

OVERDOSAGE Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS Hypersensitivity reactions such as rash, urticaria, leukopenia, agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerotomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.

DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.

HOW SUPPLIED:

Bottles of 100 Tablets	NDC 0524-0058-01
Bottles of 500 Tablets	NDC 0524-0058-05

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RU-TUSS®

Expectorant

DESCRIPTION

Each fluid ounce of Ru-Tuss Expectorant contains:

Codeine Phosphate	65.8 mg
(WARNING: MAY BE HABIT FORMING)	
Phenylephrine Hydrochloride	30 mg
Phenylpropanolamine Hydrochloride	20 mg
Pheniramine Maleate	20 mg
Pyrilamine Maleate	20 mg
Ammonium Chloride	200 mg
Alcohol	5%

Ru-Tuss Expectorant is an oral antifussive, antihistaminic, nasal decongestant and expectorant preparation.

INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief of upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms associated with hay fever, allergies, nasal congestion and cough due to the common cold.

CONTRAINDICATIONS Hypersensitivity to antihistamines. Concomitant use of an antihypertensive or antidepressant drug containing a monoamine oxidase inhibitor is contraindicated.

Ru-Tuss Expectorant is contraindicated in patients with glaucoma, bronchial asthma and in women who are pregnant.

WARNINGS Ru-Tuss Expectorant contains codeine phosphate, therefore, the patient should be warned of the potential that this drug may be habit forming. Ru-Tuss Expectorant may cause drowsiness. Patients should be warned of the possible additive effect caused by taking antihistamines with alcohol, hypnotics, sedatives and tranquilizers.

PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicle or operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease.

Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.

ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions.

DOSAGE AND ADMINISTRATION Adults: 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.

Children 6 to 12 years of age: ½ the adult dose, not to exceed 6 teaspoonfuls in any 24-hour period. Children 2 to 6 years of age: ½ teaspoonful every 4 hours, not no exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age: Use as directed by a physician.

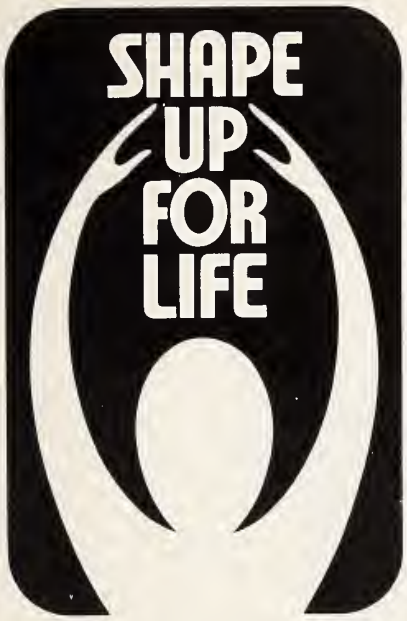
HOW SUPPLIED: (16 fl. oz.)

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AUXILIARY NEWS

An Open Letter to Kansas Physicians

We are thankful for January's slower pace that gives us a chance to recuperate from the hustle, bustle of the holiday season. Nevertheless, there is work to be done.

Doctors, we need your spouses to volunteer for the physician interview radio program. In order to put the program into operation, we need many more spouses across the state to do the interviewing. When more volunteers have been identified, we will have another training session (probably in Topeka). The program is informative and enjoyable, and Val Braun and I ask your help in soliciting the necessary personnel to implement this public relations project suggested by Dr. Heisterman to help improve the public image of the Kansas physician.

Joan Tempero (Stephen), Legislative Chairman, is planning a legislative meeting for all auxiliary members in conjunction with the mini-mid-winter board of directors meeting March 2-3 in Topeka. The Finance Committee, chaired by Phyllis Bigler (Calvin), will meet March 2 to plan the budget for next year; the special committee to study utilization of left-over convention funds, Jean Crouch (William), Chairman, will also meet that day. Plans are being made for meeting that evening with the KMS Legislative Committee for an update of legislative matters. The following day we will visit the Statehouse to attend the Senate and House sessions and possibly some committee hearings. It promises to be an interesting and worthwhile two days, and I hope you will encourage your spouse to attend.

Our Wichita chapter is hard at work on plans for the state convention April 29-May 2. I will report in more detail next month. Until then . . .

Betty L. Moore,
President
Kansas Medical Society Auxiliary

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KaMPAC MEMBERS 1982

The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

Membership Categories

Certificate	$500—C
Sustaining	100—S
Regular	60—R

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VD Hotline

The stereotype of venereal disease victims as young, nonwhite, and inner-city poor may be a thing of the past, according to a report from the Venereal Disease National Hotline. The service is operated by the American Social Health Association, and is funded primarily by the Centers for Disease Control.

Trained volunteer operators screen calls from individuals who think they have contracted a venereal disease and who do not know where to go for medical help. The VD Hotline receives more than 100,000 calls per year.

Recent statistics reveal that only 18 per cent of callers to the VD information and referral service are under 20 years old. More than one-half earn in excess of $15,000 annually, and 83 per cent are white. Additional figures show that 60 per cent of the callers are men. The mean age is 26.5 years, with nearly 30 per cent being older than 30 years. Almost 90 per cent are heterosexual, and 26 per cent are married. The mean level of education is two years of college, with 35 per cent holding a bachelor's degree. One fourth of the callers earn at least $25,000 a year.

VD Hotline officials speculate that the younger, poor, and nonwhite VD victims who no longer seem represented by the telephone data may not be calling the service because they don't know about it.

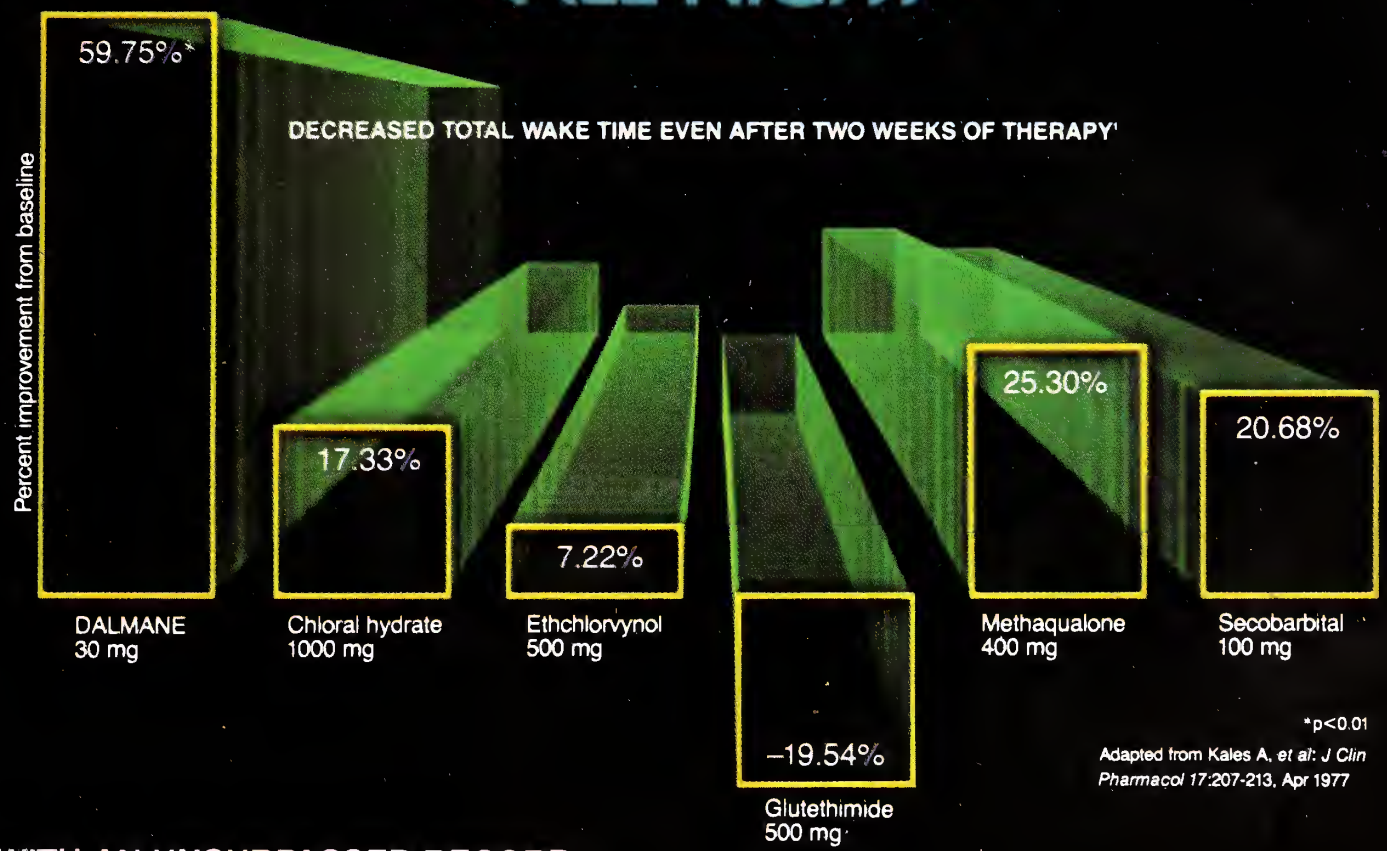
Currently, VD Hotline operators refer callers only to public VD clinics in their localities, but the telephone data indicate that many of the callers could afford and might prefer private care. Hotline officials want to assemble a nationwide corps of private physicians who could treat these middle and upper-middle class clients on a fee-for-service basis.

Physicians interested in joining this private physician referral network can contact Bea Mandel, Director, VD National Hotline, 260 Sheridan Avenue, Suite 307, Palo Alto, CA 94306. The Hotline toll-free number is 800-227-8922.

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Should insomnia recur, the patient may require guidance in setting up a regular sleep program to help provide the optimum environment for the onset of natural sleep. If hypnotic therapy is required, it should be given for the shortest time at the lowest effective dose to achieve the desired goal.

Consider other medications the patient may be taking (including alcoholic beverages) and be aware of possible drug interactions. Please note that patients should be treated for underlying physical or psychological factors before therapy with a sleep medication is undertaken.

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- No chemical interference with many commonly ordered laboratory tests, including triglycerides, uric acid, glucose, SGOT, alkaline phosphatase and total protein[5,6] (See adverse reactions section of complete product information.)
- Compatible with chronic warfarin therapy; no unacceptable fluctuation in prothrombin time reported[7,8]

UNLIKE NONSPECIFIC MEDICATIONS USED FOR SLEEP

Tricyclic antidepressants

—which are *not* sleep specific,[9] yet are sometimes used in nondepressed patients for sleep

—which can cause transient insomnia in the elderly[10]

—which can require careful monitoring in cardiovascular patients[10]

—which have strong anticholinergic effects[10]

Antihistamines

—which are *not* reliable sleep-inducing agents[11]

—which may produce stimulation instead[11]

—which have anticholinergic effects[11]

Major tranquilizers

—whose side effects may be troublesome for nonpsychotic patients[12]

—where tolerance for sedation appears rapidly[12]

Dalmane does not cause significant worsening of sleep beyond baseline levels upon discontinuation.[4]

References: **1.** Kales A, *et al*: *J Clin Pharmacol* 17:207-213, Apr 1977 **2.** Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ **3.** Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther* 21:355-361, Mar 1977 **4.** Kales A, *et al*: *Clin Pharmacol Ther* 18:356-363, Sep 1975 **5.** Moore JD, Weissman L: *J Clin Pharmacol* 16:241-244, May-Jun 1976 **6.** Spiegel HE: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ **7.** Robinson DS, Amidon EL: Interaction of benzodiazepines with warfarin in man, in *The Benzodiazepines*, edited by Garattini S, Mussini E, Randall LO. New York, Raven Press, 1973, pp 641-646 **8.** Warfarin Study: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ **9.** Baldessarini RJ: Drugs and the treatment of psychiatric disorders, chap. 19, in Goodman and Gilman's *The Pharmacological Basis of Therapeutics*, ed 6. New York, Macmillan Publishing Co Inc., 1980, pp. 391-447 **10.** Cole JO, Davis JM: Antidepressant drugs, chap. 31.2, in *Comprehensive Textbook of Psychiatry II*, edited by Freedman AM, Kaplan HI, Sadock BJ, ed 2. Baltimore, The Williams & Wilkins Company, vol 2, 1976, pp. 1941-1956 **11.** Douglas WW: Histamine and 5-hydroxytryptamine (serotonin) and their antagonists, chap. 26, in Goodman and Gilman's *The Pharmacological Basis of Therapeutics*, ed 6. New York, Macmillan Publishing Co. Inc., 1980, pp. 609-646 **12.** Davis JM, Cole JO: Antipsychotic drugs, chap 31.1, in *Comprehensive Textbook of Psychiatry/II*, edited by Freedman AM, Kaplan HI, Sadock BJ, ed 2. Baltimore, The Williams & Wilkins Company, vol 2, 1976, pp. 1921-1940

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

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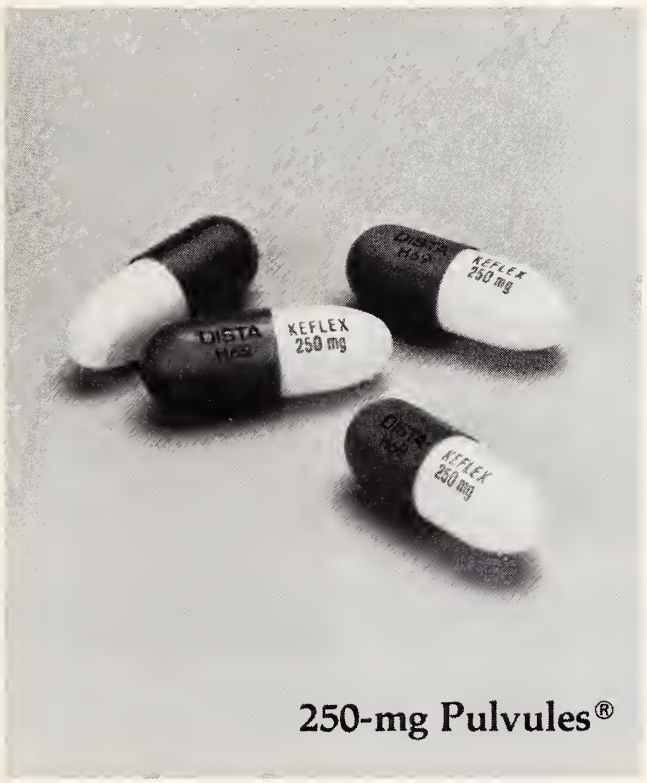
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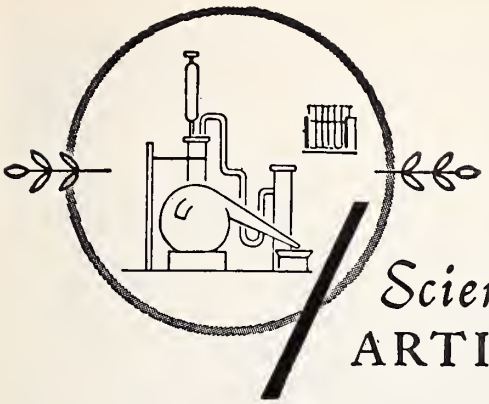
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Pharyngeal Hemorrhage

Control by Transcatheter Arterial Embolization

THOMAS H. WADDELL, M.D. *and* **TIMOTHY E. ALLEN, M.D.,** *Kansas City*

Embolization of the external carotid artery has been reported by several authors for treatment of several vascular abnormalities supplied by this artery. Dural and direct cavernous fistulas have been treated by embolization[1-4] and detachable balloon techniques.[5] The vascularity of highly vascular tumors has been reduced as a preoperative procedure in patients with meningiomas, nasopharyngeal angiofibromas, arteriovenous malformations, and glomus jugulare tumors.[5-7] Successful embolization of a fistula between the ascending pharyngeal artery and internal jugular vein has been reported.[8] To our knowledge, no cases of pharyngeal hemorrhage controlled by this method have been reported.

Case Report

A 56-year-old white female with a long history of nodular lymphocytic malignant lymphoma diagnosed in 1965 by cervical lymph node biopsy, subsequently developed involvement of the retroperitoneum, left parotid gland, trachea, and right breast. The tracheal involvement required a tracheostomy. She had received both radiation therapy and chemotherapy for control of her disease.

She presented with profuse bleeding which appeared to be pharyngeal in origin. Supportive therapy with both crystalloid solutions and whole blood were required to maintain an adequate blood pressure.

From St. Luke's Hospital, Kansas City, Missouri.
Address reprint requests to Dr. Allen, 9201 Parallel Parkway, Kansas City KS 66112.

An aortic arch and bilateral carotid arteriograms were performed. Selective right common carotid artery injection showed extravasation of contrast material into the posterior oral pharynx. Complete occlusion of the right internal carotid artery was also demonstrated *(Figure 1)*. No extravasation of contrast material occurred during the left common carotid injection.

A case of life threatening pharyngeal hemorrhage was successfully controlled by transcatheter embolization of the external carotid artery. Selective transcatheter embolization appears to be a satisfactory method of treatment for pharyngeal hemorrhage in selected cases.

Embolization of the right external carotid artery was performed following consultation with the patient's primary physician, otolaryngologist, and family. The catheter tip was positioned in the external carotid artery distal to the origin of the superior thyroid artery branch and two Gianturco coils were inserted. Injection of contrast demonstrated complete occlusion of the right external carotid artery distal to the superior thyroid artery *(Figure 2)*.

There was an immediate decrease in the rate of pharyngeal bleeding. The patient was taken to the intensive care unit where only minimal bleeding occurred during the next 24 hours. Subsequent to this, there was no further bleeding.

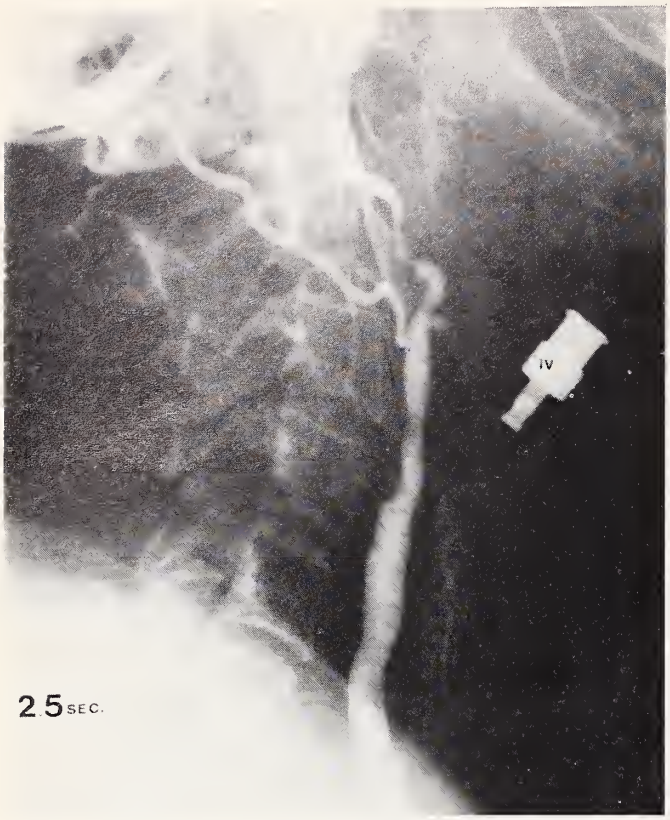


Figure 1A. 2.5 seconds after injection.

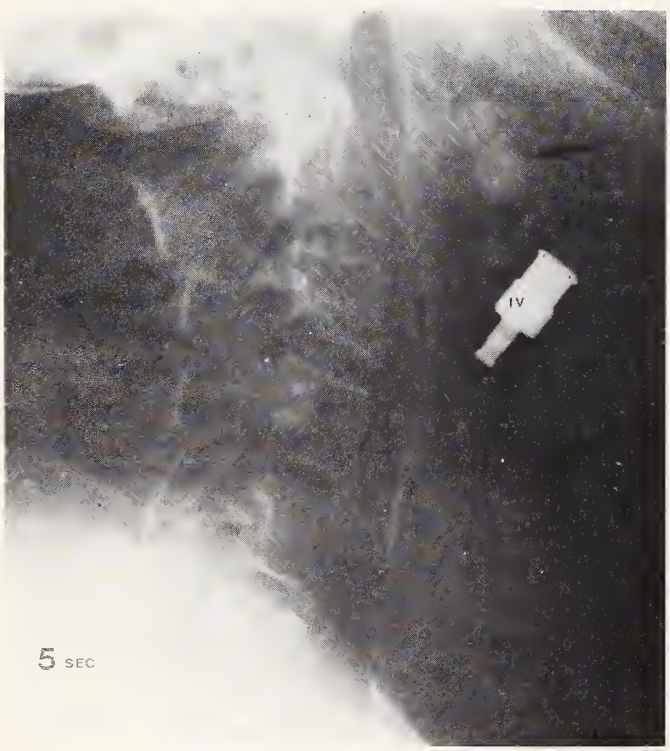


Figure 1B. 5.0 seconds after injection.

Right common carotid injection demonstrating extravasation of contrast material in the posterior oral pharynx and complete occlusion of the right internal carotid artery. (Arrow points to contrast extravasation.)



Figure 2. Right common carotid injection after embolization demonstrating complete occlusion of the right external carotid artery and no extravasation of contrast material in the oral pharynx.

The patient has shown no complication from the procedure at four months followup.

Conclusion

Embolization may be of value in controlling difficult pharyngeal bleeding problems despite the extensive collateral blood supply in this area. If this method is successful, a difficult surgical procedure may be avoided. If success is not complete, some reduction in bleeding may be obtained which could facilitate preparation of the patient for surgery and performance of the surgical procedure.

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(Continued on page 82)

Radiation Therapy Procedures

Current Trends in Radiation Therapy

RAGHUNATH P. REDDI, M.D.,* *Wichita*

About 50 per cent of all patients in whom cancer is diagnosed will receive radiation therapy sometime during the course of the disease. Therapy may be either curative or palliative, and includes relief of symptoms and shrinkage of masses.

Radiation can be used alone or in combination with a surgical procedure, chemotherapeutic agent, or both. It is important that the patient understand the nature of radiation treatment as it has side effects and the possibility of late complications or sequelae. A complete informed and formal consent permit should be obtained prior to initiation of treatment. The administration of radiation therapy is a complex process *(Table I)*.

Radiobiology

Cells die when irradiated probably because the ionizing rays damage the DNA of cell nuclei. No cell or tissue, neoplastic or normal, is inherently resistant to irradiation. This damage is most severe during certain sensitive events in the cell cycle, particularly during mitosis in one of the late growth phases (G 1) just before DNA synthesis begins. When tumor cells reproduce after the normal cell, they are likely to be destroyed during treatment. For this reason, followup treatment should not be delayed until the damaged cells have an opportunity to replace themselves.

A single large treatment radiation dose is not effective because at any given time not all tumor cells are in the phase of maximum sensitivity to different radiation. In addition, the margin between recovery of cells and necrosis of normal tissue from irradiation is narrow. The track (margin between recovery of cell and tolerance of tissue curve) widens when the treatment is protracted in overall time with the number of treatments or fractions.

Radiotherapy

The basic objective of radiotherapy is to obtain maximum control of the tumor with a minimum of complications. Optimal control rates are not the same for all types of disease, and depend upon the stage of the disease, location, organ involved, and the histopathology. The control rate of 90 per cent for early lesion is satisfactory because the sigmoid curve reduced to flatten out and increased control rate is obtained to the extent of a disproportionate incidence of complication and sequelae.

Radiation therapy may be either curative or palliative, and can be used alone or in combination with surgery or chemotherapy. Meticulous clinical evaluation, treatment planning, and therapeutic techniques yield excellent results.

Surgery and Irradiation

The combination of surgery and irradiation has been used since the early days of the century, either as preoperative or postoperative or suppurative for a primary lesion and the regional lymphatics.

The recurrence of cancer at the primary site of the surgery will demonstrate the evidence of microscopic disease present along the fascial planes, nerve sheaths and periosteum of bone, which is beyond the limits of resection. Radiation therapy can eradicate cancer at the periphery of the lesion where the extent of cancer is small and the cells are well oxygenated. However, therapy may fail in the center of large masses, primary or metastatic, that contain anoxic areas. Because of the failure of surgery or radiotherapy alone to control advanced disease, surgical procedures are frequently combined with pre- or postoperative radiation. The therapy sequence is determined by: (1) extent of the surgical procedure following pre-operative radiation therapy; (2) behavior of the cancer; and (3) the site of the tumor.

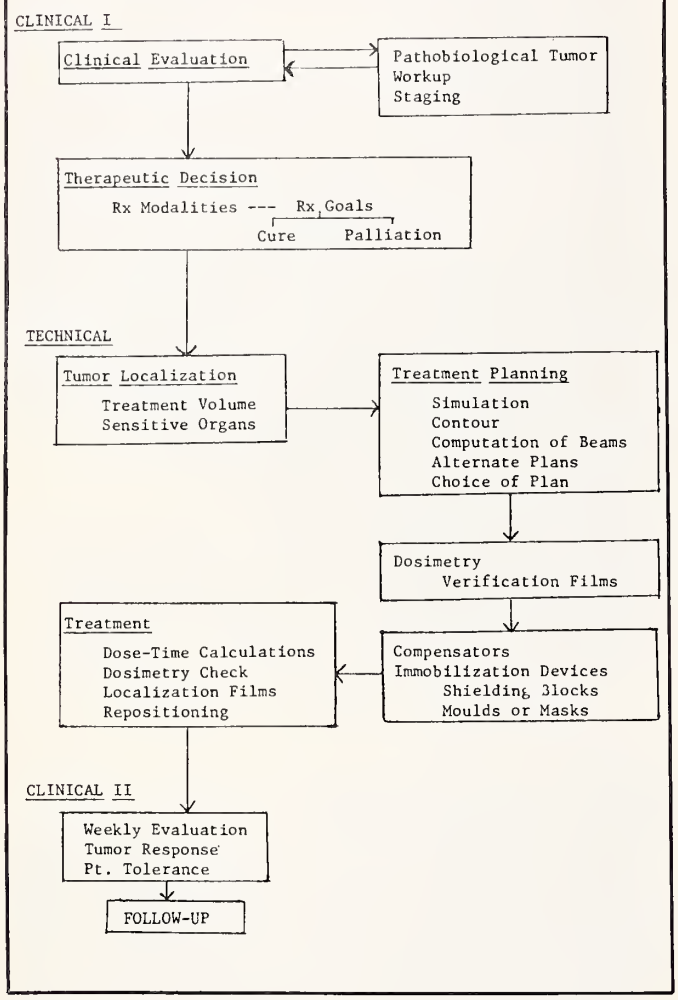
Optimum Dose

The optimum preoperative dosage of radiation has been difficult to define in terms of maximum cure rate and minimal or acceptable postoperative com-

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TABLE I
CURRENT TRENDS IN RADIATION THERAPY
RADIATION THERAPY PROCEDURES



plications; it will vary with anatomic site and cell type of cancer. Four to five thousand rads in four to five weeks has proved to be satisfactory.

Optimum Interval

Two competing factors are involved in determining an optimum interval. One involves the time necessary for normal tissue to recover from radiation damage in order that its own healing will proceed uneventfully. The other is the need to avoid cancer cell proliferation to the point where benefit from tumor shrinkage is lost.

In compliance with these criteria, it has been determined that radiation therapy may be administered at a rate of 2,000 rads in five treatments preceding immediate surgery; 3,000 rads in 15 treatments during two to three weeks; or 4,500 rads in 35 treatments during four to six weeks.

Preoperative Radiation

The purposes of preoperative radiation are:

- To eradicate subclinical disease beyond the margins of surgical resection;
- To diminish the tumor implantation that occurs in the number of viable cells in the operative field;
- To diminish the incidence of distant metastasis that occurs in viable cells entering the bloodstream at the time of surgery; and
- To convert non-resectable cancers to resectable ones.

Negative factors involved in decisions regarding preoperative radiation therapy include

- delays in wound healing;
- delays of curative surgery;
- gross shrinkage of the cancerous mass which may tempt the surgeon to perform less radical surgery possibly resulting in marginal recurrence, or cause the patient to delay or refuse resection;
- increased cost;
- interruption in the usual sequence of care; and
- a necessity for complex scheduling.

Postoperative Radiation

Postoperative radiation is indicated

- to eradicate the residual disease in the operative field;
- to eradicate new disease in adjacent areas such as the margins and in the contralateral neck;
- for all T4 lesions of head and neck cancers; or
- to decrease recurrence in positive surgically treated nodes.

The optimum time to begin postoperative radiation therapy will depend upon the healing of the surgical wound. The sooner the irradiation is administered, the more effective it is likely to be.

Radiation Reaction

Early reactions which may be noted during radiation therapy consist of erythema, dry and moist desquamation and, with continued radiation, necrosis will result. The optimum radiation reaction for preoperative patients is dry desquamation, and for postoperative patients, moist desquamation.

Depending upon the area being treated, general symptoms occasionally seen during treatment include nausea and a drop in CBC.

Late or delayed reaction depend upon

- type of treatment used — KeV, cobalt, or linear accelerator;
- total tumor dose;
- time-dose relationship; and
- total volume being irradiated.

(Continued on page 84)

Herpes Zoster

A Ten-Year Experience With Cytosine Arabinoside Treatment

H. RICHARD McFARLAND, M.D.,* *Kansas City*

ACUTE segmental herpes zoster (shingles, zona) is an infectious disease caused by varicella-zoster (V-Z) virus, and is characterized by inflammation of the dorsal root ganglia, neuralgic pain, and crops of clustered vesicles localized to dermatomes innervated by the affected ganglia. Herpes zoster results from the reactivation of a latent varicella infection.[1, 2] Depressed host resistance may be responsible for reactivation of the V-Z virus.[3-5] Cytosine arabinoside (Ara-C) is reported to inhibit deoxyribonucleic acid (DNA) synthesis in dividing cells, and is cytotoxic for certain viruses.

Since other treatment for zoster had been disappointing, administration of Ara-C began in 1971. This method of treatment was initially suggested by Kansas City hematologist, Robert Skillman, M.D. By 1973, several authorities had suggested Ara-C for systemic,[6-11] meningoencephalitic,[3, 11] and local zoster.[11, 12] At this time, Stevens *et al.*[13] conducted a controlled trial which demonstrated the adverse effects of cytosine arabinoside on disseminated zoster. Although their study dealt only with immunosuppressed patients with disseminated disease and not with spontaneous acute segmental zoster, enthusiasm for Ara-C waned. Likewise, the double blind study by Zaky *et al.*[14] concluded that Ara-C was not beneficial for localized zoster, but they administered the drug subcutaneously rather than intravenously. Subsequently, there have been other articles suggesting that systemic adenine arabinoside is beneficial[15, 16] or that topically applied idoxuridine in dimethyl sulphoxide (DMSO) is the preferred treatment for cutaneous zoster.[11, 17] Reported here is a series of 70 patients, 50 of whom received Ara-C for acute segmental zoster and 20 for chronic post herpetic neuralgia.

Subjects and Method

The patients had acute, blister stage, segmental herpes zoster and no disease thought to be associated with zoster. Most were treated as outpatients. Cytosine arabinoside (Ara-C), 50 mg dissolved in 2.5 ml bacteriostatic water for injection with benzyl alcohol 0.9 per cent weight in volume, was administered intravenously push for four to five consecutive days. Contact with the rash by clothing and medicaments was avoided. A complete blood analysis (CBC) was obtained at the beginning and end of treatment. Patients were instructed to report back if pain persisted or recurred.

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Intravenous cytosine arabinoside (Ara-C) is an effective treatment for acute segmental herpes zoster. Prompt relief of pain, rapid healing, and freedom from post herpetic neuralgia were experienced by 88 per cent of patients. Providing dermatitis has resolved, Ara-C does not significantly relieve post herpetic neuralgia.

Results

Ages of the 50 patients who received Ara-C for acute zoster ranged from 19 to 87 years; the mean age was 60 years. Females predominated 3:1, and the right side of the body was involved 3:2. The ophthalmic division of the trigeminal nerve was affected in 15 patients, the cervical area in four, the thoracic area in 25, and the lumbosacral area in six. Mean time duration prior to treatment was 5.7 days. The mean number of days of Ara-C treatment was 4.6.

Forty-four of 50 patients (88%) became and remained significantly free of pain by the fifth treatment. Only three patients were considered treatment failures due to persistence of pain in the area of dermatitis; three others experienced gradual resolution of discomfort during several weeks and were regarded as failures. One of these had received prednisone prior to the start of Ara-C treatment. The six (12%) treatment failures seem to be related to prior use of steroids or delay in treatment. The only adverse reaction was nausea and vomiting which can in part be prevented by slow injection (2-3 minutes) and relieved by trimethobenzamide HCl, tablet or

suppository. None of the patients developed a significant leukopenia or thrombocytopenia.

Twenty patients with post herpetic neuralgia were treated by this method. Ten patients (50%) had relief from chronic pain. A successful response related to earlier therapy and the persistence of activity in the area of involvement, *i.e.*, moist, raised, reddish patches in the skin. Conversely, if the skin area was dry and scars barely visible, treatment was not beneficial.

Comment

These results concern segmental zoster, not generalized or encephalitic zoster. One patient did have dissemination of lesions between the second and third day of treatment, but by the fifth infusion of Ara-C, his blisters had eschared and he was pain free. None of the patients had a disease known to be associated with zoster, *i.e.*, Hodgkin's disease, lymphoma, or varicella. All received a thorough neurologic examination and, when indicated, x-rays of the spine, and none had evidence of spinal tumor. Those with involvement of the ophthalmic division of the fifth cranial nerve frequently received ophthalmologic consultation, but due to the prompt response to Ara-C, few required topical treatment, and none had significant ocular complications. This dosage of Ara-C (50 mg) is relatively small and free of toxicity, yet effective.

Conclusion

Fifty patients with acute (blister stage) segmental herpes zoster were treated with intravenous cytosine arabinoside, 50 mg, by slow injection for four or five consecutive days. Eighty-eight per cent of patients completing the treatment course had prompt resolution of pain and blisters without post herpetic neuralgia.

Twenty patients with post herpetic neuralgia were treated with this method, and 50 per cent improved. Successful palliation occurred only when the zoster area appeared persistently irritated, and not when the area was white and dry.

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Vox Dox

Vox Dox Editor:

In my recent article, "Substance Abuse: How Physicians Recover" *(The Journal of the Kansas Medical Society,* November 1981), I overlooked mentioning Prairie View Mental Health Center of Newton as having successfully treated physicians in Kansas. This was simply an oversight on my part, as their programs have, in fact, treated a number of physicians for substance abuse for some years.

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Abdominal Scintigraphy

Locating the Site of Intestinal Bleeding

NIRANDON WONGSURAWAT, M.D.,* ***St. Louis, and*** **ADOLPH PELLEGRINI, M.D.†**
and **SALVADOR DIPALING, M.D.,‡** ***Topeka***

THE SOURCE of acute intermittent gastrointestinal bleeding can be difficult to diagnose. This case report describes detection of intestinal bleeding by abdominal scintigraphy with 99m Tc-pertechnetate.

A 59-year-old man was admitted with a complaint of bloody stools. He had been healthy until recent development of rectal bleeding. There was no diarrhea, abdominal pain, or tenesmus. Investigation by his physician, including barium enema and proctosigmoidoscopy, failed to reveal the cause or site of bleeding. On admission, results of physical examination were normal except for bloody stool detected during rectal examination. Laboratory tests included complete blood counts, urinalysis and multiple blood chemistry; results were within normal limits. Proctosigmoidoscopy up to 20 cm from the anus revealed no lesion; some clotted blood was seen in the sigmoid colon. Repeat barium enema and upper gastrointestinal series yielded negative results. The possibility of bleeding Meckel's diverticulum was raised. Abdominal scan with intravenous

Various techniques can be utilized to determine the site of intestinal bleeding. Abdominal scintigraphy with 99m Tc-pertechnetate may provide accurate results without invasive procedures or complications.

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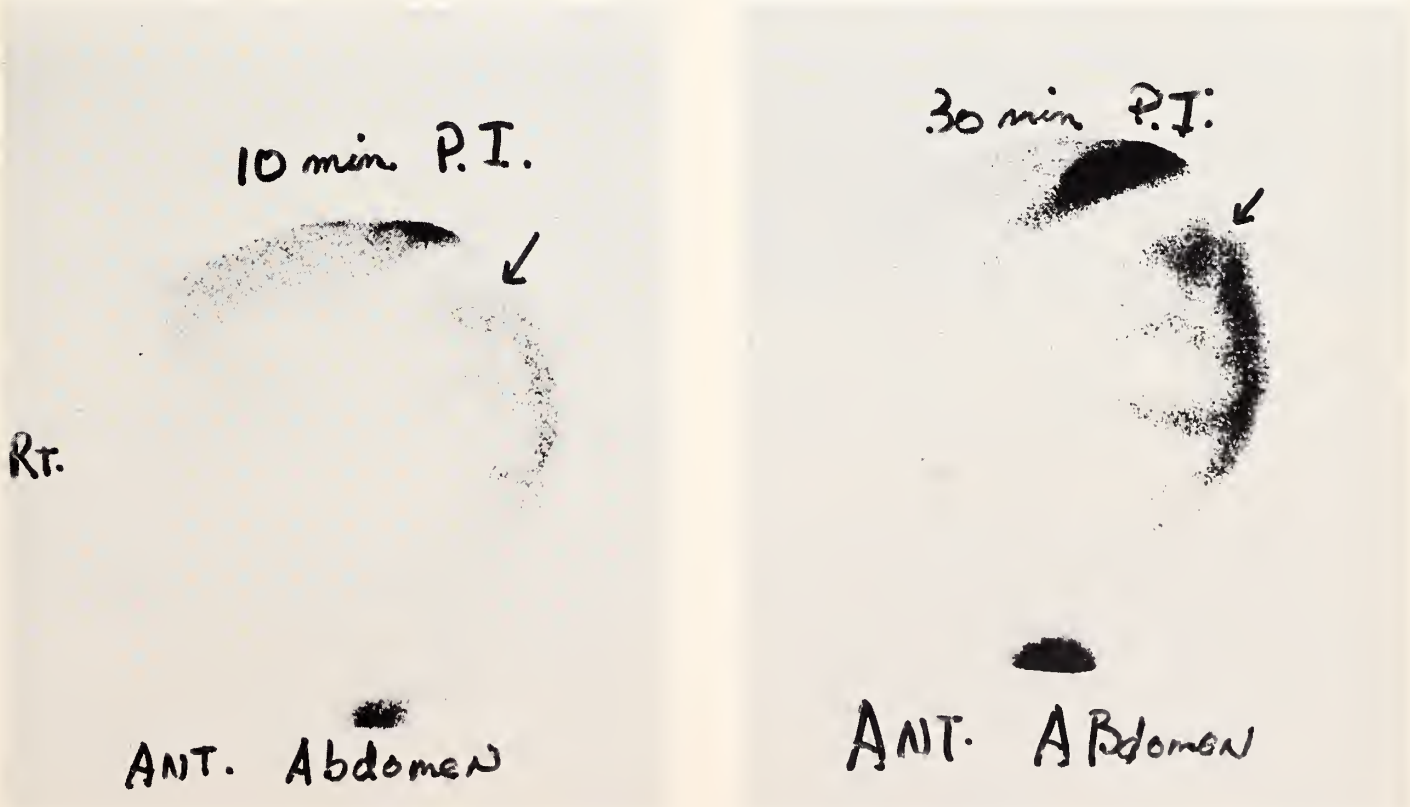


Figure 1. Collection of tracer in the descending colon (arrow) at 10 minutes (A) and 30 minutes (B) following intravenous injection of 5 mCi 99m Tc-pertechnetate.

99m Tc-pertechnetate was performed *(Figure 1)*. A noticeable amount of radionuclide was seen in the descending colon ten minutes after injection. The collection of pertechnetate was much more pronounced after 30 minutes. The patient underwent exploratory laparotomy, and a bleeding polyp, 1 cm in diameter, was found in the descending colon. A partial colectomy was carried out. The patient has had no further bloody stools since the operation.

Proctosigmoidoscopy is useful in demonstrating the lesion in many patients with rectal bleeding. However, the lesion must lie within reach of the scope. The average depth of insertion was 19.5 cm.[1] Conventional radiographic techniques, such as barium studies, fail to demonstrate the source of bleeding in 20-30 per cent of all patients, and if a lesion is visualized, there is no certainty that it is the bleeding site.[2, 3] Angiography is an excellent technique, but it is invasive, has the potential to cause serious complications, requires well-trained personnel to perform it properly, and shows hemorrhage only at the time of bleeding.[4, 5]

The alimentary canal is not a good subject for tracer imaging. Nuclear techniques have been disappointing in locating the site of intestinal bleeding. One exception is the case of Meckel's diverticulum. This ectopic hemorrhage can be extremely difficult to diagnose and is frequently missed by angiography. 99m Tc-pertechnetate is taken up by parietal cells, which are frequently present in a Meckel's diverticulum; thereby the existence of the abnormality is documented. In our patient, however, the lesion was a polyp. There were no gastric parietal cells to take up the tracer. The puddling seen in the descending colon soon after intravenous injection is the tracer in the plasma compartment which leaked into the bowel lumen from the bleeding polyp.

Acknowledgement

Stan Bechtold provided technical assistance for this paper.

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THE 34th ANNUAL MIDWEST CANCER CONFERENCE

March 19-20, 1982
The Hilton at East Kellogg & Rock Road
Wichita, Kansas

ONCOLOGY OVERVIEW 1982

FRIDAY, MARCH 19

Early Detection — Moderator, Hewitt Goodpasture, M.D., Wichita

8:00- 8:15 Registration
8:15- 8:30 Welcome — Paul Baumann, M.D., President; Jay Zatzkin, M.D., Program Director
8:30- 9:10 Early Detection of Colon Cancer: Marsha Liepmann, M.D., Assistant Professor, University of Michigan Medical Center
9:10- 9:50 Early Detection of Lung Cancer: Richard Wheeler, M.D., Assistant Professor, University of Michigan Medical Center
9:50-10:10 BREAK
10:10-10:50 Early Detection of Cervical Cancer: Richard Wheeler, M.D., Assistant Professor, Univ. of Michigan Medical Center
10:50-11:30 Early Detection of Breast Cancer: Frances E. Bull, M.D., Professor of Internal Medicine, Univ. of Michigan Hospital
11:30-12:00 Panel

State of the Art in Oncology Today — Moderator, David Street, M.D., Wichita

1:30- 2:20 State of the Art in Surgical Oncology: Dr. A. W. Boddie, Jr., Associate Surgeon, M. D. Anderson, Univ. of Texas
2:20- 2:30 BREAK
2:30- 3:20 State of the Art in Radiation Oncology: Dr. Luther Brady, Jr., Professor and Chairman, Hahnemann Medical College, Philadelphia, Pennsylvania
3:20- 3:30 BREAK
3:30- 4:20 State of the Art in Medical Oncology: David L. Ahmann, M.D., Professor, Mayo Medical School
4:20- 4:50 Panel

SATURDAY, MARCH 20

Supportive Care — Moderator, David Rosen, M.D., Wichita

8:30- 9:10 Nutritional Support: Marsha Liepmann, M.D., Assistant Professor, University of Michigan Medical Center
9:10- 9:50 Pain Control: Frances E. Bull, M.D., Professor of Internal Medicine, University of Michigan Hospital
9:50-10:10 BREAK
10:10-10:50 Ethics of Informed Consent: Marsha Liepmann, M.D., Assistant Professor, University of Michigan Medical Center
10:50-11:35 Role of Oncology Nurse: Barbara Medvec, R.N., Hematology-Oncology Outpatient University of Michigan Medical Center
11:35-12:00 Panel

Prostatic Carcinoma

Efficacy of Interstitial Radiotherapy

RAGHUNATH P. REDDI, M.D.,* *Wichita*

In the 1930s, external radiation therapy was employed in the treatment of patients with advanced carcinoma of the prostate. This technique was virtually abandoned in the 1940s in favor of hormonal therapy after the discovery by Huggins and Hodges of the hormone dependence of many prostatic neoplasms. External irradiation emerged again in the 1950s, largely because of the advent of megavoltage equipment such as cobalt-60 teletherapy units, linear accelerators, and betatrons.[1] These machines permitted easy delivery of high doses of gamma and x-radiation to deep-seated tumors without the limitation of severe skin reactions imposed by orthovoltage machines. This improvement in technology has led to a shift in the philosophy of treatment, from the use of radiation for the relief of pain and obstruction in advanced disease to the definitive treatment of relatively well localized earlier cancer. Enough experience has been accumulated to demonstrate that prostatic carcinoma can be successfully treated by external irradiation.[2-5] In recent years great interest has been shown also in assessing the role of interstitial irradiation in the curative management of this neoplasm.[6-10]

Prostatic carcinoma, which has a very slow growth rate and remains localized for long periods, seems suitable for treatment with interstitial implantation. Encapsulated radioactive iodine^{125} has been recommended for the treatment of early stage prostatic cancers[11] for the following reasons: (1) localization is more precise and the distribution can be adapted to an irregular tumor shape; (2) the dose received by the bladder and the rectum is 25-30 per cent of that delivered to the tumor; and (3) treatment time is much shorter. Furthermore, the amount of damage to peri-prostatic tissue has been minimal. Iodine^{125}, a more recently developed radionuclide, offers additional advantages.[6-8] This radioisotope emits gamma rays of low energy (27KV) so that radiation exposure is minimal. Film badges worn by personnel in the operating room while performing these implants record no significant radiation, and the patient does not require isolation following the procedure.

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Presented at the American Society of Therapeutic Radiologists, 21st Annual Meeting, October 1979, New Orleans, Louisiana.

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External radiation therapy has proved safe and effective in achieving local control in the treatment of carcinoma of the prostate. The superiority of interstitial irradiation, as compared to external beam radiotherapy, has been demonstrated in the management of readily accessible tumors.

The purpose of this paper is to review and present our experience of biopsy findings, complications, and survival following treatment of prostatic cancer with iodine^{125}

Material and Technique

All patients with carcinoma limited to the prostate were eligible for this procedure between July 1975 and June 1978. All had been diagnosed by needle biopsy as having carcinoma of the prostate and were appropriately evaluated. Pre-implant evaluation included physical examination, chest films, bone scan (skeletal survey, if indicated), blood urea, serum alkaline and acid phosphatase, intravenous pyelogram, and bone marrow aspiration. A lymphangiogram was optional and only five patients underwent this procedure, all with negative results. All 39 patients underwent bone marrow aspiration and biopsy prior to implant from the posterior iliac crest. All specimens were negative for metastatic disease.

Thirty-two patients were found to have stage B and seven patients were stage C with no elevation of acid phosphatase. Thirty-one patients were white and eight were black. The youngest patient was 49 years and the oldest was 83 years with a median age of 68 years. Thirty-three (85%) patients *(Table I)* presented with one or more symptoms which included nocturia, diminished stream, frequency and

TABLE I
ST. LOUIS UNIVERSITY MEDICAL CENTER
(1975-1978)

CA of Prostate		
Asymptomatic	6	15%
Symptomatic	33	85%
Nocturia		32
Diminished stream		28
Frequency & hesitancy		24
Bladder neck obstruction		13
Dysuria		6
Hematuria		3
Pain		1

hesitancy of micturition, bladder neck obstruction, dysuria, hematuria, and pelvic pain. The remaining 15 per cent of patients were asymptomatic.

Impotence occurred in a substantial number of patients; *i.e.*, 19 of 39 patients prior to the implant. All 20 patients who were potent retained potency following therapy. However, 25 per cent of these patients have complained of failure to ejaculate, but all had transurethral resection (TUR) prior to implant, and it seems likely that they have retrograde ejaculation secondary to the operation.[12]

The retropubic approach was used to allow for better assessment of the extent of the disease, especially the status of the pelvic lymph nodes, and to facilitate the insertion of radioactive sources in the prostate. A bilateral total or partial extraperitoneal pelvic lymph node dissection was performed and clips were placed at the level of the nodal dissection. This facilitated planning for supplemental external irradiation if the pelvic nodes proved to be positive for metastasis.

After completion of node dissection hollow stainless steel 17 gauge needles (15 cm long) were inserted in the prostate gland, spaced approximately 1.0-1.5 cm apart. The posterior extent of the gland (the depth of insertion of the needles) was determined by digital rectal examination and by a Foley catheter in the urethra.

The iodine^{125} seeds were then inserted through the needles. The needles were withdrawn in fixed increments as calculated earlier so that the seeds were deposited in the gland in a lattice pattern with regular spacing between seeds. After completion of the procedure the incision was closed leaving a Penrose drain in the retropubic space. Using a stereoscopic shift, radiographs were obtained to identify the location of the iodine seeds. A computer program was used to calculate the dose distribution. The doses within the prostate ranged from 20,000 to 30,000 rads, delivered during one year. The dose decreased very rapidly to 5 per cent at 2.0 cm. About two-thirds of the total dose was delivered during the first four months.

The number of seeds implanted in each patient varied from 13 to 44, with a median of 28 seeds with a calculated dose of 16,000 rads at 0.5 cm from the implant volume, based on post-implant nomographs. The individual seed activities for clinical applications typically varied in the range of 0.58-0.75 millicuries. Half-life of iodine^{125} seeds is 60 days. In all but two cases, position of the implants within the prostatic tissue was considered good.

Results

Thirty-nine permanent implants were carried out between July 1975 and June 1978 at St. Louis University Hospital *(Table I)*. The tumors were graded histologically as to degree of differentiation. In 33 patients, they were found to be moderately differentiated, and in six patients, poorly differentiated, which revealed an absence of or only occasional glandular pattern. Thirteen of 39 patients had TUR for bladder neck obstruction with 5-30 gm of prostatic tissue excised.

Lymph Node Metastasis

Of 39 patients, seven underwent total extraperitoneal pelvic lymphadenectomy, which included the excision of bilateral hypogastric and obturator nodes. Lymphatic metastases were found at the time of implant in six patients (15.5%). Four of 32 patients (12.5%) had stage B tumor, and two of seven patients (28.6%) had stage C disease, four of six patients (67%) with poorly differentiated tumors had lymph node involvement compared to only two of 33 patients (6%) with well differentiated tumors. The degree of differentiation of the primary tumor had a definite correlation with the incidence of nodal metastases.[13-16]

External Radiation Therapy

Of six patients with positive lymph nodes, five received external radiation therapy. All five were treated with 10 MV linear accelerator with four ports (''Box Technique'') and custom lead blocks were made to protect the prostate (10,000 rads isodose line) from AP, PA, and lateral ports. A tumor dose of 6,000 rads was delivered to pelvic lymph nodes in 33 treatments with daily increments of 180 rads with all ports being treated daily. Incidence of diarrhea,

TABLE II
ST. LOUIS UNIVERSITY MEDICAL CENTER

I^{125} Seeds Implant
Minor Complications

Pt	*Age*	*Seeds*	*Ext Rt*	*Complications*
BJ	70	34	no	*WH — 90 days
BS	50	21	no	*WH — 28 days
BK	68	39	no	*WH — 30 days
RJ	73	32	no	*WH — died on — 57th Day — pulmonary embolism
OD	76	35	6,000 rads	Dysuria frequency — 6 months
DM	72	26	no	Rt pelvic pain — 12 months IVP + CT — NAD

* Wound healing.

TABLE III
ST. LOUIS UNIVERSITY MEDICAL CENTER
(1975-1978)

I^{125} Seeds Implant
Moderate to Severe Complications

Pt	*Age*	*Seeds*	*Surgery + RT*	*Complications*
DJ	57	20	Surg + 6,000 rads	† (S) edema of scrotum & legs
WC	59	32	6,000 rads	‡ (M) edema of scrotum & legs
HJ	62	26	Surg + 6,000 rads	** (S) edema of scrotum & left leg
HE	72	22	*IH no	proctitis — 12 mos biopsy negative
LJ	60	33	6,000 rads	proctitis — 24 mos biopsy negative radiation changes

* Internal hemorrhoids.
† Severe.
‡ Moderate.

dysuria, and frequency of micturition was minimal and did not warrant interruption of treatments.

Morbidity

In this study, morbidity was categorized as early or late as well as the relationship of symptoms to surgery, external radiation therapy, and urinary or gastrointestinal tracts.

Early Effects

No intra-operative complications were seen in our series during the surgical operation or implantation of the seeds. Six patients developed early complications which were minor. However, on the 57th day, one of the six patients died of pulmonary embolism, which was related to deep vein thrombophlebitis of the lower extremity. Minor wound infection limited to anterior to the rectus fascia was seen in three patients; this required prolonged hospital stay for incision, drainage, and healing *(Table II)*. One patient developed a dull constant pain in the right paravascular area which persisted for a year. IVP and CT scan of the pelvis yielded negative results and the pain spontaneously resolved in 12 months. Biopsy of the prostate was also negative at the end of one year. The sixth patient acquired severe dysuria and frequency of micturition which lasted for six months following therapy. This patient had positive nodes in the pelvis and received external radiation therapy. The dysuria was of an aseptic nature and was satisfactorily controlled by urinary antispasmodics.

Late Effects

Three of five patients *(Table III)* had late complications of moderate to severe scrotal and leg edema which started nine months following the external irradiation; all of these patients had undergone one or more previous operations such as bilateral hernia repair, appendectomy, or heart surgery with the venous graft being taken from the femoral region. In addition, all three patients received external radiation therapy. It appears that these complications were related to prior operations in the groin and pelvic lymphadenectomy, aggravated by the external radiation therapy. The remaining two patients acquired severe proctitis six months later which persisted 12-24 months. One had internal hemorrhoids that might have resulted from the radiation but no radiation changes were noted on biopsy. The second patient received external radiation, and biopsy of the rectum revealed radiation changes. It is likely that the severe proctitis in the patient was due mainly to external irradiation. Local control was defined in this group of patients as complete regression of the palpable prostatic nodule as determined by rectal examination during the followup period ranging from four to twelve months. Local failure was defined as nodular or diffuse induration persisting or reappearing during the same period of time.

Followup

Two of the 39 patients were lost to followup. Three patients died — one due to pulmonary embo-

TABLE IV
ST. LOUIS UNIVERSITY MEDICAL CENTER

CA of Prostate — 39
(Followup)

Pulmonary embolism	1	— died 57th day
Lost to followup	1	
Evaluable Patients	37	
Local control		35 (94.5%)
Partial response		2 (5.5*)

1 died — coronary heart disease — 14 months
1 died — diffuse mets — 25 months

lism; one at 14 months due to coronary heart disease; and one of diffuse metastases at 25 months *(Table IV)*.

Thirty-seven patients were available for evaluation at one year, and 22 were studied for two years or more. The local control rate in 37 patients was 94.5 per cent with 35 patients undergoing biopsies at the end of the year.

Biopsy

Followup needle biopsies from two different sites were done at intervals of approximately 12 and 24 months. Post-treatment biopsies were compared with pre-treatment or initial diagnostic biopsies. Biopsy results were classified in three groups:

- *Negative pathology report:* Evidence of vascular dysplasia and significant stromal changes. These changes included small artery hyalinization, fibrosis, fat necrosis, and symplastic giant cells.
- *Indeterminate pathology report:* The pathologist was unable to make a definite statement that excluded residual viable tumor. There was some evidence of cytoplasmic swelling and stromal changes which was similar in appearance to the pretreatment biopsy specimen.

TABLE V
ST. LOUIS UNIVERSITY MEDICAL CENTER
(1975-1978)

I^{125} Seeds Implant
Biopsy Results: 35 Patients
at 1 yr

Negative	20/35	57%
Indeterminate	6/35	17%
Positive	9/35	26%

TABLE VI
ST. LOUIS UNIVERSITY MEDICAL CENTER
(1975-1978)

I^{125} Seeds Implant
Biopsy Results: 9 Patients
two yrs

Indeterminate	5	2-N, 1-I, 2-P
Positive	4	2-N, 1-I, I-P

N — Negative.
I — Indeterminate.
P — Positive.

- *Positive pathology report:* Definite evidence of persistent residual tumor consistent with the original biopsy specimen.

The biopsy reports were tabulated on 35 patients at the end of one year *(Table V)*: Twenty (57%) were negative; six (17%) were indeterminate; and nine (20%) were positive. Twenty-two patients were followed for two years or more, but only nine *(Table VI)* — five indeterminate and four positive — were biopsied, and results were negative in four, indeterminate in two, and positive in three patients. Those with negative biopsies at the end of one year were not subjected to further biopsy procedures. In summary, at the end of two years *(Table VII)*, biopsies for 24 of 35 patients were negative (68.5%); three (8.5%) were indeterminate; and eight (23%) were positive.

Discussion

This is the first reported series of subsequent needle biopsy of patients with adenocarcinoma of the

TABLE VII
ST. LOUIS UNIVERSITY MEDICAL CENTER
(1975-1978)

I^{125} Seed Implant
Biopsy Results: 35 patients
(summary)
3 yrs followup

Negative	24/35	68.5%
Indeterminate	3/35	8.5%
Positive	8/35	23.0%

prostate previously treated by interstitial radioactive iodine^{125} seed implants. It is a small series followed for a minimum period of three years. In interstitial irradiation, the total dose is of great significance.[6-8] Our study of cancer of the prostate shows 100 per cent clinical local control if a minimum of 16,000-20,000 rads can be delivered to the prostate. When total dose is less, local control decreases[16] to two-thirds or one-half that with higher doses. There is considerable clinical and laboratory data available to indicate that radiation response of adenocarcinoma is the same as that of other epithelial tumors, except slower.[9, 11, 16] In this study the percentage of patients with indeterminate (17%) and residual (26%) tumor reports initially changed to 8.5 per cent and 23 per cent respectively after one year. At the end of two years, however, the presence of demonstrable tumor in 23 per cent of patients is somewhat alarming. Hilaris[16] reported that in his series of 112 patients, local failures occurred in only 10 per cent of patients with prostatic implant and pelvic lymph node dissections, but only selected and suspicious patients were biopsied. Grout and associates reported positive prostatic biopsies in five of 11 patients more than six months after completion of radiation therapy. Bagshaw[2] reported a series of 49 patients in which only five had biopsy, and three of these revealed the presence of viable tumor. Cox[12] reported that in 30 patients, 12-30 months after external irradiation, 20 of 25 were negative. Kagan *et al.*[17] reported that nine of 18 patients had positive biopsies at one year, nine of 13 had positive biopsies at two years, and three of six had positive biopsies at three years. Seventeen of 20 patients had a negative clinical examination at two to seven years. The three patients with clinical recurrence had positive biopsies post-radiation, but it does not appear that a positive biopsy can always be associated with clinical regrowth.

Post external irradiation biopsies demonstrate the slow regression rate of prostatic cancer and the insignificance of biopsy results at least during the first year after irradiation.[17, 18] The significance of subsequent biopsies as well as the volume and stage of the disease, time-dose factors, and possible hormone manipulation remains to be seen.[18]

Conclusion

Iodine^{125} seed implantation has far less morbidity when another modality such as external irradiation is not added.[16] The ultimate role of I^{125} implantation in the management of localized prostatic cancer is yet to be determined. Early experiences with this technique are consistent with the possibility that it will prove to be as effective as total prostatectomy or external radiation therapy in terms of patient survival. Present analysis suggests that it may prove superior to these alternatives in terms of the quality of survival.

Additional external radiation therapy to the pelvic and para-aortic node areas neither prolong life or the disease-free interval nor alter the pattern of distant dissemination,[16] but it increased the morbidity to 100 per cent in our group of patients.

Evidence of lymph node involvement was lower than might be anticipated,[16] but most patients had partial lymphadenectomy and were also a very selected group.

The main advantages of iodine^{125} seed implant and pelvic lymphadenectomy as compared to other modalities are: (1) accurate staging; (2) favorable local control; (3) satisfactory survival; (4) low morbidity and mortality rates; (5) short hospital stay in most instances; (6) accurate high dose adapted to tumor size and shape; and more importantly, (7) preservation of the functional integrity of pelvic organs.

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Acoustic Tumors

Surgical Update in 42 Consecutive Patients

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EVALUATION of our recent series of 42 consecutive patients operated upon for acoustic tumor removal has resulted in some changes in medical and surgical management. We are convinced that the benefits of the translabyrinthine approach are superior to any other approach for tumor removal.

Pantopaque cisternography has been virtually eliminated with the use of the third generation C-T scanner. We feel it is the single most important diagnostic test for acoustic tumors. Auditory brainstem response (ABR) has become the most accurate audiometric test. However, patients with hearing loss may exhibit wave V and interwave latencies deceiving to the inexperienced eye.

The C-T scan has assumed a particularly dominant role in the exclusion of acoustic tumors. This, coupled with a high index of suspicion, remains a clinical cornerstone in the diagnosis of acoustic tumors.

Case Material

We have incorporated our 20 previously published cases[1] with the additional 22 cases and listed them in *Tables I* and *II* in consecutive order. The patients have been divided into two groups — those with and those without displacement of the IVth ventricle.

We have not changed our original criteria for facial function from the previous report. To reiterate, + + + indicates normal facial function with no minimal defect; + + less than normal facial function but complete eye closure, with or without some synkinesis, and normal appearance in repose; + some movement of facial musculature but not acceptable as a good result, and incomplete eye closure although normal facial appearance in repose. The tables indicate both long and short term results in all patients at the time of this writing. Some patients with + + designation will become + + + with the passage of time. In addition, we expect two patients who have facial paralysis to regain function. Both of these patients had anatomical continuity of the VII nerve at the completion of the operation. One patient had the facial nerve resected without repair. Two patients had the facial nerve re-routed with end-to-end (VII-VII) neuroanastomosis. Both have + facial function, with one patient eight months postoperative. Twenty-two patients awakened on the operating table with absolutely normal facial function. We consider this a significant benefit of translabyrinthine removal, a result not achieved by suboccipital surgery.

A series of 20 consecutive patients who underwent operation for removal of acoustic tumors has been previously reported. During the subsequent 12-month period, an additional 22 patients were operated consecutively. This report updates our surgical experience in the total series of 42 patients.

Fewer panopaque cisternograms were done with identification of smaller tumors on the third generation C-T scanner. We feel it is important for reporting purposes that tumors be measured in millimeters or centimeters and noted as with or without displacement of the IV ventricle. In our series, an occasional outside scan did not have a cursored measurement and required a fixed scale measurement to determine its size.

Results

We accomplished total tumor removal, microscopically, in all patients except for Case 42 which was a planned, elective, partial (95%) tumor removal. This patient was 77 years of age. The tumor was removed entirely from the brain stem, and a small portion was left on the facial nerve as it entered the internal auditory canal. There were no fatalities. Complete loss of facial function occurred in only three patients and there were no increased neurologic deficits other than cranial nerves VII and VIII.

Sponsored by the Midwest Ear Research Institute, 2945 Wyandotte Street, Kansas City, MO 64108.

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TABLE I
CORRELATION OF VII NERVE FUNCTION WITH NON-DISPLACEMENT OF IV VENTRICLE ON C-T SCAN

Case	*Age*	*Pantopaque*	*C-T*	*VII Function*	*Surgery*
1	52	+	—	+ + +	Oct. 76
3	58	+	—	+ + +	May 77
8	46	+	—	+ +	Mar. 78
9	52	+	2.0 cm.	+	June 78
10	65	+	—	+ + +	Aug. 78
12	63	+	—	+ + +	Jan. 79
13	35	+	—	+ +	Feb. 79
17	63	not done	1.6 cm.	+ + +	Feb. 80
18	29	not done	0.9 cm.	+ + +	Mar. 80
20	35	not done	0.45 cm.	+ + +	Apr. 80
21	49	not done	2.5 cm.	+ + +	May 80
22	48	not done	1.36 cm.	+ + +	July 80
24	57	not done	1.8 cm.	+	Aug. 80
25	52	+	IAC	+ + +	Aug. 80
27	42	not done	2.0 cm.	+ + +	Oct. 80
28	58	not done	1.5 cm.	+ + +	Nov. 80
29	62	Memphis	0.5 cm.	+ + +	Nov. 80
30	66	not done	1.5 cm.	0	Dec. 80
31*	40	not done	1.0 cm.	+ +	Dec. 80
32	62	+	0.9 cm.	+ +	Jan. 81
33	53	not done	1.0 cm.	+ + +	Jan. 81
34	60	not done	1.0 cm.	+ + +	Feb. 81
35	65	St. Louis	2.0 cm.	+ + +	Mar. 81
36	63	not done	1.6 cm.	+ + +	Mar. 81

* Facial paralysis seven years previously with + + residual, presumably Bell's palsy.
Acoustic tumors — N = 42.

TABLE II
CORRELATION OF VII NERVE FUNCTION WITH DISPLACEMENT OF IV VENTRICLE ON C-T SCAN

Case	*Age*	*Tumor Size*	*VII Function*	*Surgery*
2	36	1.8 cm.	+ + +	Jan. 77
4*	56	3.5 cm.	0	Dec. 77
5	68	3.2 cm.	+ +	Sep. 77
6‡	57	3.0 cm.	+	Oct. 77
7	51	3.2 cm.	+ +	Nov. 77
11	21	2.5 cm.	+ + +	Jan. 79
14	55	2.0 cm.	+ + +	Feb. 79
15	54	1.7 cm.	+ +	Nov. 79
16*	26	3.8 cm.	+ + +	Jan. 80
19	64	2.5 cm.	+ +	Mar. 80
23	42	3.0 cm.	+ + +	July 80
26‡	32	3.0 cm.	+	Oct. 80
37	39	3.0 cm.	0	Mar. 81
38	40	3.9 cm.	+ + +	Mar. 81
39	23	3.0 cm.	+ + +	Apr. 81
40	35	3.6 cm.	+ +	Apr. 81
41	63	2.5 cm.	+	May 81
42†	77	3.5 cm.	+ + +	June 81

* Pre-operative hydrocephalus.
† Elective 95% partial removal.
‡ Rerouting of VII nerve and (VII-VII) neuroanastomosis.
Acoustic tumors — N = 42.

TABLE III
LONG AND SHORT TERM VII
NERVE FUNCTION RESULTS

Tumor Size	*Proportion With Weakness*	*Percentage With Weakness*
IV Ventricle midline	6/24*	25.0%
IV Ventricle displaced	8/18	44.4%
Total	14/42	33.3%

* One patient had Bell's Palsy seven years prior to tumor removal with some residual weakness.
Acoustic tumors — N = 42.

We preserved useful hearing in Case 25 through a middle cranial fossa approach. An attempt to preserve hearing through the suboccipital approach was unsuccessful in Case 8. Our 31st patient acquired a presumed Bell's palsy seven years prior to operation with slight residual weakness. Facial function postoperatively was unchanged.

The results in our series compare favorably with others regarding total tumor removal, fatality, and facial function *(Tables III, IV, V).*

Discussion

Our most troublesome complication has been postoperative CSF rhinorrhea (Cases 10, 11, and 21), and CSF wound leak (Cases 24 and 40), requiring secondary closure of the wound. Since Case 21, we have opened the facial recess and obliterated the middle ear and eustachian tube in all cases.[2] We have not had subsequent CSF rhinorrhea requiring reclosure of the wound. Three patients (Cases 25, 26, and 30) had postoperative psychoses of undetermined cause. One patient required transfer to the psychiatric unit. These patients were all receiving dexamethasone, 4 mg, and cimitidine, 300 mg, every six hours pre- and postoperatively.

TABLE IV
LONG AND SHORT TERM VII
NERVE FUNCTION RESULTS

Tumor Size	*Proportion With Paralysis*	*Percentage With Paralysis*
IV Ventrical midline	1/24	4.2%
IV Ventricle displaced	2/18	11.1%
Total	3/42	7.1%

Acoustic tumors — N = 42.

Beginning in July of 1980, we have administered prophylactic antibiotics to all patients, and none have developed meningitis since that time. In January 1981, we began administering steroids to all patients regardless of tumor size.

We have devised an operative technique for very large acoustic tumors and meningiomas called a one-stage transtentorial, retrosigmoid, translabyrinthine approach. Utilizing this approach, the tentorium is opened and the superior petrosal sinus ligated and divided. The translabyrinthine approach is completed without opening the dura. The retrosigmoid dura is exposed and opened. The tumor is gutted extensively from behind and above, and then removed in standard translabyrinthine fashion.

This approach was used in two patients, one with a large meningioma, with preservation of facial function in both cases. We were prepared to use this approach in Cases 37, 38 and 39, but found a routine translabyrinthine approach quite adequate for exposure. Because of tight space in Cases 40 and 41, extradural middle fossa retraction with a Yasargil retractor and Silverstein sigmoid retractor was helpful.

Long and short term results of facial nerve function are shown in *Tables III* and *IV*. Our series is compared to others in *Table V;* our results compare favorably with those of other surgeons.

Review of our cases shows that the translabyrinthine approach was used exclusively and without embellishment in 35 of the 42 cases.

Conclusion

Our combined experience with the translabyrinthine approach has convinced us that this method is superior to the old suboccipital approach with regard to postoperative facial function. We are now able to report our results in 42 cases. We feel that surgeons

(Continued on page 82)

TABLE V
SUMMARY OF RESULTS IN 42 CONSECUTIVE
OPERATIONS FOR ACOUSTIC TUMOR REMOVAL

	No. Cases	*Total Removal*	*Mortality*	*VII Paralysis*
Luetje Whittaker	42	97.6%	0%	7.1%
House, *et al.*[3] Ditullo Malakasian	500	93.4%	2.8%	13.5%
Rand (1978)[4]	79	91.0%	3.7%	12.0%

Acoustic tumors — N = 42.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

An Update on Pneumococcal, Influenza, and Hepatitis Type B Vaccines

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ACTIVE immunization remains one of the most effective means in the prevention of infectious diseases. Although routine immunization procedures have been an important part of medical practice in pediatric care, all physicians should be cognizant of up to date information in various vaccine developments. This paper reviews the current status of three immunizing agents: pneumococcal polysaccharide, influenza virus, and hepatitis virus B vaccines.

Pneumococcal Polyvalent Polysaccharide Vaccine

The Problem. Pneumonia is the fifth most common cause of death in the United States and is a leading cause of morbidity and mortality in the world population. It is estimated that there are 200,000 to one million cases of pneumococcal pneumonia with 13,000-66,000 deaths in the United States annually.

The Vaccine. Streptococcus pneumoniae is a gram positive coccus possessing a capsule made of polysaccharide polymers. The capsular polysaccharide is the virulent factor for pneumococcus and will evoke type specific protective antibody in patients' sera. There are more than 80 capsular types of pneumococci; only 14 of these (U.S. nomenclature: types 1, 2, 3, 4, 6, 8, 9, 12, 14, 19, 23, 25, 51, and 56) account for more than 80 per cent of bacteremic pneumococcal diseases.[1] The current vaccine is composed of 50 μg/ml of each of these 14 types of capsular polysaccharide in a 0.5 ml solution. After field trials showing that the current vaccine would protect 75-95 per cent of normal adults from pneumococcal pneumonia, the vaccine was approved by the Food and Drug Administration in February 1978.

Candidates for Vaccination. The vaccine is not intended for general use. Patients who should receive the vaccine include the following: (1) persons with chronic underlying diseases, such as chronic obstructive pulmonary disease, various kinds of heart diseases with congestive failure, severe diabetes, renal failure, and alcoholics with cirrhosis of the liver; (2) patients having splenectomy or splenic dysfunction, such as homozygous sickle cell disease; and (3) elderly patients in chronic care facilities.

Although early trials on pneumococcal vaccine showed good antibody response two to three weeks after immunization in normal adults, children under 2 years of age do not respond well because of their very low serum level of IgG-2.[2] Other investigations revealed certain patient populations having ineffective or delayed antibody responses. In patients with Hodgkin's disease receiving combined therapy of irradiation and chemotherapy, the antibody response was poor; however, if the vaccine was given seven to ten days before initiation of treatment, the antibody response was not depressed.[3] Patients with chronic renal failure undergoing hemodialysis showed a lower antibody response during the first month of immunization. However, six months after vaccination, the geometric mean antibody concentration as measured by radioimmunoassay in hemodialysis and

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control subjects were more nearly alike.[4] Sickle cell disease leads to functional asplenism and interferes with phagocytosis and intracellular killing of microorganisms. Increase of opsonization antibody by vaccination in these patients or in postsplenectomized individuals should help to provide protection from fulminating pneumococcal infections.

Contraindications and Side Effects. Children two years of age or younger and those individuals showing hypersensitivity to any components of the vaccine are contraindicated as recipients of the pneumococcal vaccine. Post-immunized immunity lasts for at least five years and may be longer. About a third of vaccine recipients develop local redness and soreness over injection site for one or two days, and 3 per cent of them may have transient low grade fever. Because of a marked increase in adverse reactions with reinjection of pneumococcal vaccine, second or "booster" doses should *not* be given at this time.

The pneumococcal vaccine can be given simultaneously with the influenza vaccine at different injection sites without altering the immune responses of either vaccine.

Influenza Vaccine

The Problem. There are three antigenic types of influenza viruses: A, B, and C. Distinct subtypes are found in influenza A viruses and, to a lesser extent, in type B viruses. Influenza C virus does not seem to cause epidemics and no subtypes are known.

Both influenza A and B viruses, particularly the former, give rise to recurrent epidemics of febrile respiratory infections every one to three years contributing to excessive deaths. The most devastating influenza A epidemic of 1918-1919 resulted in 20 million deaths throughout the world with 500,000 deaths in the United States alone. During an epidemic, the attack rate may reach 10-40 per cent of the population involved.

The Vaccine. Influenza virus has a central helix containing eight segments of single stranded RNA genome within a lipoprotein envelope. The surface projections contain hemagglutinin (H) and neuraminidase (N), which undergo periodic changes accounting for the various subtypes or strains of influenza A viruses.[5]

With the passage of time there may be major antigenic changes in the prevalent strains, and the U.S. Public Health Service regularly reviews the antigenic characteristics of strains of influenza viruses in order to select the proper viruses to be included in the formulation of contemporary vaccine.

Although both live-attenuated and inactivated influenza viral vaccines have been developed, only the inactivated vaccine is licensed in this country. The inactivated vaccine is prepared from influenza virus grown in the allantoic sac of chick embryos. The infected allantoic fluid viruses are inactivated with formaldehyde and are concentrated and purified to remove impurities and chick proteins.

Whole virus and split (by ultrasonization) virus vaccines are manufactured. The split virus vaccine has been associated with fewer side effects, but is also less antigenic. The 1980-81 trivalent vaccine contained 7 μg of purified antigens from three strains of influenza viruses: A/Bangkok/79 (H_3N_2), A/Brazil/78 (H_1N_1), and B/Singapore/79. The 1981-82 influenza vaccine has been recommended by the U.S. Public Health Service to include the same three strains of influenza viruses, but to have the antigenic strengths increased to 15 μg per strain in each dose.[6]

The efficacy of the vaccine is about 70 per cent, and the duration of induced immunity is about six months. Annual administration of the influenza vaccine given before the month of November is, therefore, necessary to insure protective immunity.

Candidates for Vaccination. Debilitated patients, the elderly (especially those over age 65), institutionalized individuals, and patients with chronic pulmonary, cardiovascular, renal, or metabolic disorders should receive the influenza vaccine. Health and public service personnel, such as policemen, firefighters, and municipal government employees have next highest vaccination priority.

The inactivated influenza virus vaccine comes in 0.5 ml per dose to be given intramuscularly or subcutaneously *(Table I).* For people who were born before 1952, in which they had experienced "Russian flu" (H_1N_1), one dose is sufficient. For those who were born after 1952 and have not received influenza vaccine in previous years, two doses, given four weeks apart, are recommended. For patients with cancer and on maintenance chemotherapy, the efficacy of vaccination is only 30-35 per cent. A full immune response (approaching that for normal adults) could be elicited by giving the vaccine once chemotherapy has been stopped for more than 30 days.[7]

Side Effects and Contraindications. Side reactions from influenza vaccinations are few. The following reactions have been reported:

1. Local redness and induration at the site of injection may occur in less than one-third of vaccinees, and lasts for one to two days.
2. Toxic systemic symptoms, such as fever,

TABLE I
RECOMMENDED 1981-82 INFLUENZA VACCINE DOSAGE ACCORDING TO AGE

Age Group	*Vaccine Preparation*	*Dosage ml*	*# of Doses*
29 yrs and older	Whole virus or split virus	0.5	1
13-28 years	Whole virus or split virus	0.5	2*
3-12 years	Split virus	0.5	2*
6-36 months	Split virus	0.25	2*

* Given four weeks apart between doses, two doses are essential to assure good protection unless the individual had received at least one dose of influenza vaccine between 1978-1980. In such instances, one dose is sufficient.

myalgia, and malaise lasting one to two days may occur in 1-2 per cent of adult vaccinees. These systemic symptoms are more commonly seen in children, and may be reduced by using a split virus vaccine.

3. Immediate allergic reactions are extremely rare, but may occur in individuals who are allergic to egg protein. Persons with a history of anaphylaxis to egg protein should not receive influenza vaccine.

4. Prior to 1976, Guillain-Barré syndrome (GBS) was not reported to be associated with influenza vaccination. During the 1976 national immunization program against swine influenza, there was a statistically significant excess risk in persons developing GBS within ten weeks following vaccination with A/New Jersey/76 ($H_{sw1}N_1$) influenza vaccine. The incidence was 1/100,000 in vaccinated persons, about five to six times higher than in unvaccinated individuals. Since 1978, active surveillance was adopted by the Center for Disease Control,[8] but no clear association between influenza vaccination and GBS was uncovered. The reason for the excess risk of GBS following the swine influenza strain vaccination remains unknown.

Because influenza vaccine is inactivated, there is no evidence to suggest it would carry any maternal or fetal risk. For its use in pregnancy, the physician should evaluate the pregnant woman's need for influenza immunization on the same basis used for other individuals as mentioned above.

Amantadine hydrochloride (Symmetril), an antiviral agent, has been shown to have protective value against influenza A viruses by prevention of penetration of virus into host cells; it is not effective for influenza B viruses. It is not a substitute for influenza vaccination, but may play a supplementary role during an influenza epidemic for high risk unvaccinated individuals. The drug must be taken daily by those unvaccinated persons during the epidemic period. Amantadine does not interfere with active immunization of influenza vaccination. It may be given to unvaccinated individuals during an epidemic at the time influenza vaccine is given and be continued for 10-14 days until immunity from active immunization is developed.

Hepatitis B Vaccine

The Problem. Hepatitis B virus (HBV) infection is a worldwide problem. The incidence of chronic hepatitis B surface antigen (HBsAg) carriers in certain geographic areas, such as Taiwan, may range from 10-20 per cent. In the United States, the incidence is 0.2-0.5 per cent. However, a very high incidence may be found in certain populations in the United States, such as male homosexuals and patients or medical staff in hemodialysis units. The possibility that hepatocellular carcinoma may be casually related to HBV infection is substantiated by the fact that as many as 80 per cent of the patients with hepatoma have HBsAg in their blood and most of them have post-necrotic cirrhosis of the liver.[9]

The Vaccine. HBV is a DNA virus containing several distinct antigens — HBsAg, HBcAg, and HBeAg. In chronic carriers, the surface antigen HBsAg may reach a very high concentration, 10^{12} or 10^{13} particles per ml of blood. The hepatitis B vaccine is prepared by purification of HBsAg from positive human serum by ultracentrifugation or by affinity chromatography and inactivated by 1/2000 to 1/4000 formalin.[10] The vaccine has been under extensive investigation for years and was recently approved by the FDA for clinical use.

Efficacy of HBV Vaccine. Maupas and associates vaccinated patients and medical staff in a hemodialysis unit in France with hepatitis B vaccine as a pilot study.[11] Only 5.6 per cent of vaccinated staff members and 3.0 per cent of vaccinated patients showed subsequent HBV infection compared to 50 and 87 per cent infection rates respectively in their unvaccinated counterparts. Szmuness and col-

TABLE II
COMPARISON OF PNEUMOCOCCAL, INFLUENZA, AND HEPATITIS B VACCINES

	Pneumococcal	*Influenza*	*Hepatitis B*
Nature of immunizing agent	Capsular polysaccharide	Inactive virus	Particles of HBsAg
Composition	Polyvalent, 14 types	Trivalent (Types A & B)	Monovalent or bivalent (Subtypes of HBsAg)
Cross protection	Absent	Absent	Present between subtypes
Efficacy	75-95%	70%	96%
Duration of immunity	5 years	6 months	Several years ?

leagues reported a placebo-controlled randomized double-blind vaccination study in 1,083 homosexual men known to be at high risk for HBV infection.[12] All participants received either HBV vaccine or placebo. Within the first two months, 77 per cent of the participants receiving the vaccine developed high serum antibody levels against HBsAg. When a booster dose was given a month later, the rate of significant antibody level increased to 96 per cent. During the first 18 months after vaccination, clinical and subclinical HBV infection developed in only 1.4-3.4 per cent of vaccinated individuals, compared with 18-27 per cent in placebo recipients ($P < 0.0001$). None of the vaccinees with a detectable immune response had clinical hepatitis B infection or asymptomatic antigenemia.

The vaccine was found to be safe and the incidence of side effects was low. Only 16 per cent of vaccinees had soreness over injection sites; (10.6% in placebo group) and 2.6 per cent had fever (2.2% with placebo), which was not statistically significant.

Vaccine Candidates. Persons at high risk of HBV infection may be selected for vaccination. These would include residents and staff in mental retardation institutions and patients and medical staff in hemodialysis units, physicians, dentists, clinical laboratory personnel, spouses and newborn babies of HBsAg carriers, and possibly travelers to endemic areas. Control of HBV infection should prevent acute and chronic hepatitis, a high percentage of post-necrotic cirrhosis of liver and hepatocellular carcinoma. The estimated cost of the HBV vaccine is $75.00-$125.00 for a series of three injections.

Summary

An updated review of pneumococcal, influenza, and hepatitis B vaccines is presented *(Table II)*. In this review, the problems of these illnesses, compositions of the immunizing agents, indication and contraindications, and effectiveness as well as adverse effects are discussed. Physicians and public health administrators should take the responsibility for encouraging people at risk to be vaccinated.

Self-Assessment Questions

1. The pneumococcal vaccine is made of 14 serotypes of killed pneumococci and may prevent more than 90 per cent of bacteremic pneumococcal infection. (T or F)

2. If the pneumococcal vaccine is given 7-10 days before chemotherapy of Hodgkin's disease, the vaccine can achieve the immune response comparable to normal individuals. (T or F)

3. The current 1981-82 influenza vaccine consists of H_3N_2, H_1N_1, and $H_{sw1}N_1$ strains of influenza viruses. (T or F)

4. Although the Guillain-Barré syndrome was seen in approximately 1/100,000 vaccinees during the national program of influenza immunization in 1976, the recent surveillance of the current vaccine without swine influenza virus suggests that there is essentially no direct relationship between influenza vaccine and Guillain-Barré syndrome. (T or F)

5. The hepatitis B virus vaccine is made from purified HBsAg and has been found to be a safe immunizing agent. (T or F)

(Answers on page 78)



Drug INFORMATION

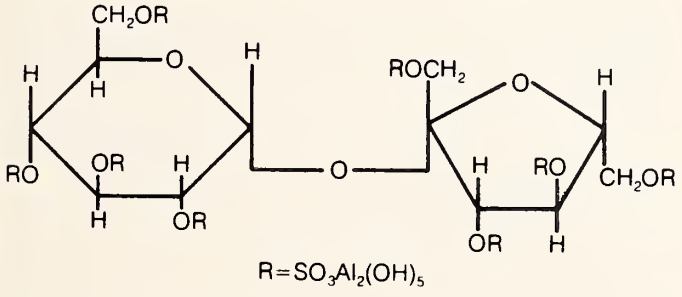
LINDA HOGAN, M.S., R.Ph., *Editor*

Sucralfate

Question: Can you briefly explain the indications and mode of action for the new drug, sucralfate?

Answer: Sucralfate (Carafate®-Marion Laboratories) is a new, nonsystemic, anti-ulcer drug that is currently indicated for the short term treatment of duodenal ulcers.

Sucralfate is an aluminum salt of a sulfated disaccharide that forms a highly polar anion when put into solution. This high polarity is responsible for its poor gastrointestinal absorption and relatively non-



R=$SO_3Al_2(OH)_5$

systemic action.[1] The small amounts of the sulfated disaccharide that are absorbed are excreted primarily in the urine. Although the exact mechanism of action is not yet fully udnerstood, clinical studies suggest that sucralfate's anti-ulcer activity is the result of formation of an ulcer-adherent complex that preferentially binds to the ulcer site and protects it from further destruction by acids, pepsin, and bile salts.[2] Human studies have shown that therapeutic doses of sucralfate do not have antacid effect in the gastrointestinal tract.[3] Antacids may be added to sucralfate therapy to relieve pain associated with ulcers, but like antacids, sucralfate should probably not be given concurrently with tetracycline.[4] Other studies comparing cimetidine and sucralfate have indicated no significant difference in the rate or extent of ulcer healing between these two anti-ulcer drugs.[5]

Because of its minimal systemic absorption, observed adverse reactions to sucralfate have been relatively few (4.7%). Constipation has been the most frequently reported complaint at a rate of 2.2 per cent of patients.[4]

Sucralfate is available in 1-gram tablets and is competitively priced with cimetidine on a cost-per-day basis. Current dosing guidelines recommend a 1-gram tablet to be taken four times daily on an empty stomach for up to eight weeks of therapy.

References available from the Drug Information Service, UKSM-KC, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

The Drug Information Service is maintained by the Department of Pharmacy at the K.U. Medical Center to promote rational drug therapy. The Service is staffed by clinically oriented pharmacists with access to accurate, current, and unbiased information. Information is available free of charge to any health care practitioner involved with patient care. Contact:

Drug Information Service
3930 Cambridge
Kansas City, KS 66103

Telephone (any time): 913-588-2328

The President's Message



What lies ahead?

Two hundred years ago only 20 per cent of the people lived to age 65 years. Now only 20 per cent of the people do *not* live to age 65. In 1900 there were more than 20 people for every person over 65, while today that figure is less than ten to one. In another half century the figure should halve again to about five to one.

Clearly, the support base for the elderly is shrinking rapidly. On the other hand the health care costs for the older generation are increasing astronomically. It is not only a matter of rising expenses because of increasing numbers, but also the escalating costs of care for each older individual. Good health care (especially pediatric) was the key to improving longevity statistics cited above; now it is also part of the problem that we see before us.

Health care for the elderly is expensive and will become increasingly more so as labor, inflation, and energy costs continue to climb and the number of people using these health services continues to rise. Also, as technology becomes more sophisticated, it will be possible to prolong life somewhat but at staggering economic consequences. Some say that today more than 50 per cent of an average individual's entire lifetime medical expenditure occurs during the last ten days of his/her life.

Are we seeing forces headed on a collision course? Will we see some triage (unfortunately, and in all probability, based on chronological age), some form of rationing, or worst of all, health care based on "who you are"?

Dr. Cloud, President of the AMA, recently said, "cost effectiveness means 'Is it worth it?' "

What lies ahead?

Fraternally,

Herman W. Hesterman M.D.

President



Economy Class

It didn't really rate as a classical border-ruffian invasion but it did originate east of the line dividing the two neighboring — and usually neighborly — commonwealths. It seems one of the urban medical centers (those are hypertrophied hospitals which have taken on a lot of other things beside hospitalling and had to come up with a covering title) in Kansas City (Missouri, need we say?) signed up several hospitals in smaller Kansas communities to provide them with administrative and technical assistance. As it happened, the cries of alarm came not from the signees (who were apparently well satisfied with the arrangement) but from several similar centers on this side of State Line Road. They saw, not without some justice, the maneuver as an effort to man the switches and shunt innocent Kansans (and their dollars) into the Kansas City institution (and its coffers). The Kansas City spokesman predictably denied any ulterior motive so we can presume that any Kansas patients found at their doors will be given short shrift (Rover Boys expression in keeping with the spirit of this dissertation).

In spite of the implications of professional ravishment, however, the situation should be kept in perspective. First, as already implied, community hospitals that have grown to a certain size (through population growth plus cadging patients from other hospitals) have found it beneficial, even necessary, to expand their services in many directions which has meant a formidable outlay for space, equipment, and personnel. Next, each step has had a generational effect, giving rise to additional subdivisions of purpose and producing areas of service which, once established, are considered essential and indispensable (check thesaurus under "bureaucracy"). Not the least part of the expanded format is a public relations service charged with getting media notice as frequently as possible and in excess of that obtained by any other institutions in the community.

Whether by purpose or coincidence, this development has been associated with the change in emphasis from medical care to health care and the avowed dedication to all forms of community life. The stated intents are laudable: not just the guardianship of physical health but emotional as well; not just the correction of illness and injury but the prevention thereof; not just the care of the patient but the support of family and friends, too. The line separating those services that were formerly considered the valid function of the hospital from those traditionally obtained elsewhere — or not at all — is no longer at issue. Health care embraces all and hospitals are nominating themselves as the appropriate social segment to provide them, tending to ignore the contribution to the burgeoning costs of hospital sojourns — or that rates applying to all patients must cover them regardless of whether or not a given patient uses them.

But our purpose is to examine the situation, not criticize the performance. "Perspective" means placing the phenomenon in relationship to the total social and professional milieu, not whether it is right or wrong, good or bad, necessary or unnecessary. The situation has evolved because the public has accepted it as having something of value. A considerable portion of the population of this state is served by more modest institutions limited in developmental potential by population and economic realities. The full panoply of services deemed essential today is available, of course, by referral to the centers by their physicians (who have long been acquainted with the possibility of losing control of the patient to others). State lines and institutional obligations aside, the situation is affecting the character of health service as much as the more frequently cited technologic and academic advances.

Hospitals traditionally — and despite sometimes hardheaded boards — have not promoted business

practices that would please Wall Street so the adoption of techniques more compatible with current affairs is probably overdue. But this outline of the development of medical centers as they proceed toward corporate maturation doesn't do justice to some of the operational hormones that see them through organizational adolescence. It may not be evident to those not gifted with our fiscal astigmatism but there are, in fact, two rather prevalent commercial effectors at work, and they are of particular interest in these times of concern over health care costs. Obviously, an ever-increasing number of clients is necessary if the institution is going to grow. The conservative and uninitiated attitude would be that this requires a risk of capital and while there is some truth to this, it overlooks the fact that the most effective way to promote expansion is to call it economizing. (This term may not be recognizable to some of the younger ones but they may have heard it spoken of as cost containment. Check thesaurus under "euphemism.") It works like this. It costs them something — quite a bit, in fact — to provide their basic services to a given group. By enlarging the group, they can increase the number of individuals provided with the services at something less per head than it took before, so this represents a saving of the difference (attested to by a process known as cost analysis — don't bother with the thesaurus). As long as they have made the start, they can just as well do some other things which take advantage of some of the services already set up and reduce the unit overhead (and improving, it is hoped, the profit margin a little more). And since profits are slender, even in non-profit institutions, anything that may enhance them is attractive and meets with the approval of the Board. This procreational process naturally strains the existing facilities and it becomes obvious that the availability of more space will actually be an economy as it will be more efficient — and may even lead to the development of a plans and development department just to determine how efficiently things can be done. As the hospital functions extend and become more complicated and sophisticated, it is essential (not less so because others are doing it) to computerize, which is an economy because of the saving in costs for personnel even though the personnel to run the system (and help straighten out the errors) may be more expensive then their commonplace predecessors. The institution is now ripe for the computer manufacturer's sales pitch that it should have its own setup rather than buy from others — more economical.

You've been under the impression all this time that these things have been done in the name of expansion of patient service when it has actually been an exercise in economizing. Each step has been taken not with the intent of making more money — there are no greedy people in the health care world — but only in the name of efficient use of facilities to take advantage of all possible economies and keep the cost to the consumer-patient as low as possible. In a way it is symbolized by the little sack of items each patient is presented with on arrival at the hospital — the little bar of soap, the small tube of tooth paste, a noncommital brush, tissue, mouth wash, a cheery note of greeting from the management. Whatever his need for these things, he gets them because it is more economical for the hospital to buy the things already put up than to have the personnel bothered with getting them piecemeal or calling the patient's family to bring them.

By their example, however, the medical centers may be sowing the seeds of their own destruction. As they invade the outback bringing the benefits of their services, there is always the risk that some susceptible small-hospital administrator will be sensitized. It occurs to him or her one day that if they had their own scanner or whatever, they could save money. This could even lead to upgrading the laundry or getting the newfangled food carts. And if the Board will buy that, it may be ready to hear the pitch for the new south wing — and they're off and running.

If economizing is the stuff of organizational growth, competition becomes its sustenance. Physicians may have been repelled by the concept of the commercialized type of competition — unprofessional at least, damaging to the ethical posture that medical care must not be compromised. It has always been present but in a subtler and professionally more acceptable form. It comes into the medical lexicon now because, like economizing, public pressure has made it not only acceptable but desirable. It is easier to embrace it in these days when there are no longer any individual practitioners of medicine — there are associations, corporations, groups, clinics, services — even centers. The depersonalization of medical service has brought the subtler forms of competition into bolder relief — while the profession hastens to reassure the public it is all for the benefit of the purse without sacrifice of the body — and tussles with the new ethics.

So economizing and competition are essential facts in the growth of organizations. If our friends in Missouri are engaged in a little friendly competition, state lines are not the issue. They play by the same rules here and the next move is up to the competitors. — *D.E.G.*

RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
Robert P. Hudson, M.D., Kansas City ... (913) 588-7040
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
Max Teare, M.D., Garden City (316) 276-7689
David L. Trudeau, M.D., Wichita (316) 685-1111
Donald R. Tucker, M.D., Topeka (913) 354-5275
Virginia L. Tucker, M.D., Lawrence (913) 862-9360 Ext. 215
Kermit Wedel, M.D., Minneapolis (913) 392-2144

Kansas Medical Society, Topeka (913) 235-2383/235-3619

An Update on Vaccines

(Continued from page 72)

Answers

1. *False*
2. *True*
3. *False*
4. *True*
5. *True*

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MONTH IN WASHINGTON

Review of Disabilities Cases

The Social Security Administration has launched a sweeping review of the 4 million people receiving disability benefits. As many as 150,000 people may be dropped from the program this fiscal year. Social Security has alerted physicians to expect "a substantial increase in requests for medical reports."

The increase in requests for special consultative examinations "will be even more substantial because the majority of the beneficiaries will not have seen their own physician recently," Social Security said.

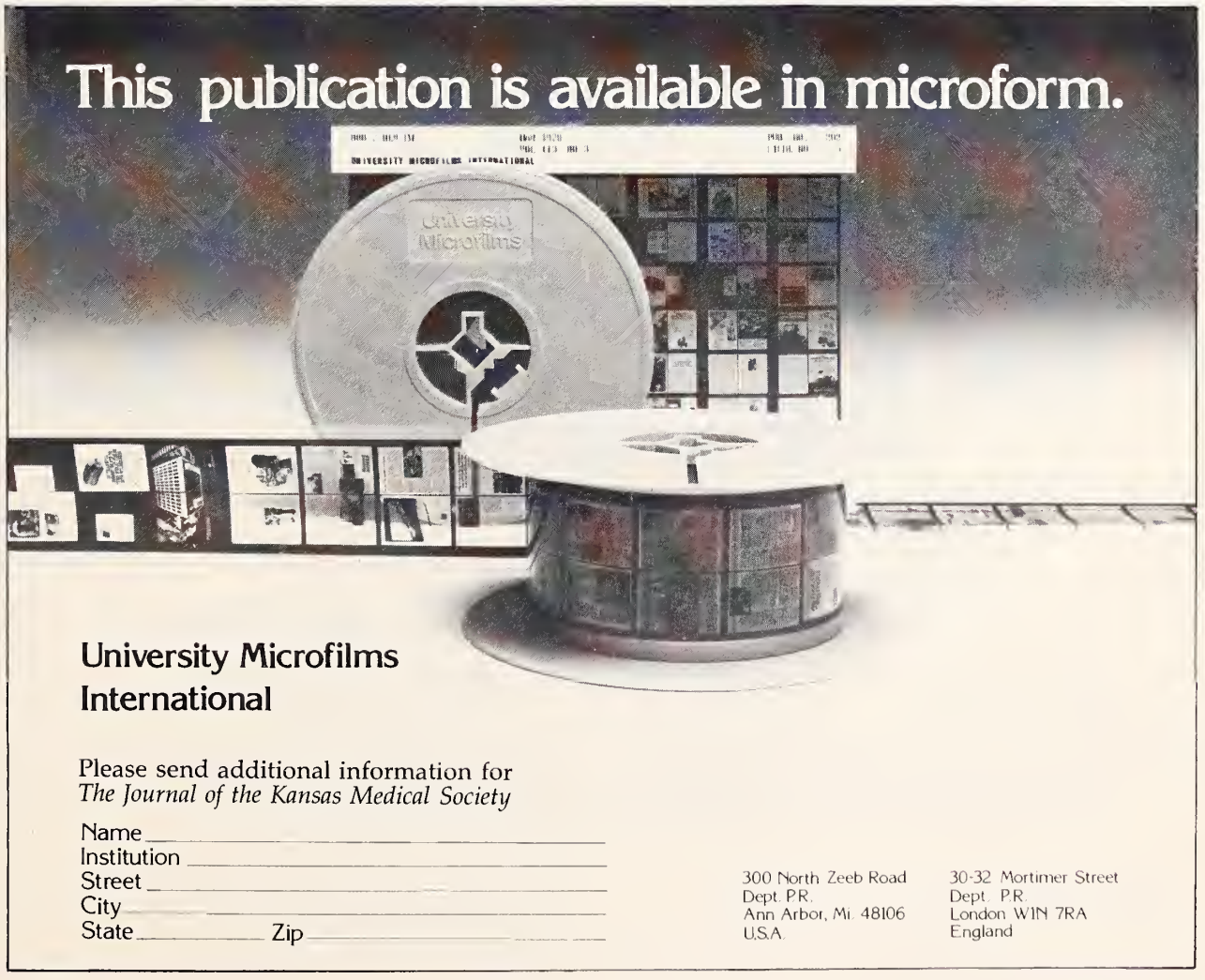
Additional physician consultants will be recruited by the State Disability Determination Services to handle the increased periodic review workload. More physicians to handle the consultative examinations will be needed.

The crackdown results from a 1980 Congressional law requiring Social Security starting this January to review nearly every disabled case at least once every three years. The basic purpose is to determine whether the beneficiary is currently unable to work.

Noting that beneficiaries may express concern to their physicians about the reviews, Social Security urged physicians to point out to their patients that they, the physicians, do not give an opinion or participate in the decision on whether the patients are disabled. These decisions are made only by the State Disability offices.

"Over the past few years, Social Security disability benefits came to be regarded by some beneficiaries as a permanent, lifetime entitlement — that benefits would continue until they retired, died, or chose to go back to work," said Social Security. "So there is going to be a lot of unhappiness and confusion for the next two to three years as disability benefits are terminated for thousands of Americans."

Emphasized the federal agency: "Social Security disability benefits, in most cases, are intended as a temporary help until people can overcome their impairment and return to work." The benefits in the future will be used "only for those who are really



unable to work because of a medically determinable disability — as the law intends.''

The review of more than 500,000 claims this fiscal year has important implications for physicians, Social Security said, listing them as:

- The beneficiary's treating physician will be asked to furnish an updated report showing the patient's current condition.
- Where information from the treating sources is insufficient to make a new determination of disability, a consultative examination will be arranged to get the needed data.
- Some patients are going to be upset about having to, in effect, reestablish their entitlement to disability benefits.
- In addition to state offices, Social Security's regional offices and headquarters will be looking for additional medical consultants. The agency estimates it will be spending almost $240 million during the next fiscal year on payments for medical evidence of record and consultant examinations, compared with $138 million during the last fiscal year and $190 million in the current fiscal year. More than 800,000 cases are slated for review in the fiscal year that starts October 1982.

Social Security issued some tips for physicians with disabled patients:

- Patients may call soon after receiving notice that their claims are to be reviewed. If the physicians have been seeing the patients regularly and have complete information about the impairments, they should advise the patients they will send in the medical reports promptly. The patients should be reminded that the physicians do not make the disability determination.
- If patients have not been seen recently, physicians may suggest a prompt, thorough examination.
- Patients may call physicians after notification they are not disabled within the meaning of the law. If physicians have already submitted a recent medical report, they should note that they had no part in the finding. But if a medical report for the review was not submitted, and the patient wishes a report, the physicians may want to conduct a special examination, since such beneficiaries have ten days to submit information for the government to reconsider its decisions.

Legal Decisions

The Supreme Court this term is slated to fix im-

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portant boundaries on the application of the federal antitrust laws to medicine.

Four cases have been accepted for review so far. The latest involve a Blue Shield plan's exclusion of payments for psychologists and the practice of peer review by physicians' committees of insurance reimbursement claims.

Previously, the Supreme Court had agreed to rule on the American Medical Association's challenge to the Federal Trade Commission's action against ethical advertising codes and the question of maximum fee schedules for medical foundations.

The decisions on these cases are not expected until this Spring.

The Blue Shield case involved two Virginia plans that excluded payment for clinical psychologists. The Fourth U.S. Circuit Court of Appeals upheld the suit against the plans charging a violation of antitrust laws.

If the high court's ruling is broad enough, it could have an impact on the question of whether health insurance can limit mental health benefits to treatment by psychiatrists or those acting under psychiatrists' directions.

The high court will also consider a case by a New York chiropractor who argued that the peer review process by insurance companies operated to set an upper limit on fees, a practice he charged is price-fixing and thus a restraint of trade.

Home Health Care

Support is picking up in Congress for legislation to expand Medicaid and Medicare coverage for home health services.

The measure's sponsor, Senator Orrin Hatch (R-UT), Chairman of the Senate Labor and Human Resources Committee, is driving for Congressional passage by year's end.

The legislation (S.234) encourages establishment of home health programs and provides tax breaks for families who care for their elderly at home.

At the Committee's final hearing on the bill before marking it up for Committee action, Hatch won support from Chairman Claude Pepper (D-FL) of the House Aging Committee who said he would introduce a companion measure in the House. A number of Senators are co-sponsors of Hatch's bill.

The Senator said more than 25 million Americans are over 65 and that three-fourths of them may be forced to enter nursing homes instead of staying with their families. The Congressional Budget Office said some 1.7-2.7 million people could be in need of expanded home service, but that less than 500,000 people receive it. Less than 2 per cent of Medicare outlays — $750 million — go for home health care.

Surgeon General

The long ordeal of C. Everett Koop, M.D., is over. The Senate has confirmed his nomination to be Surgeon General by a vote of 68-24, ending a seven-month controversy centering around Dr. Koop's outspoken views against abortion.

The nomination was stalled because it was necessary to grant a congressional waiver to allow Dr. Koop to take the post because of age and Public Health Service experience requirements in law. This allowed opponents, led by Chairman Henry Waxman (D-CA) of the House Commerce Subcommittee on Health, to block Senate waivers.

The issue was always abortion, however. Senator Orrin Hatch (R-UT), Chairman of the Senate Labor and Human Resources Committee, told the Senate that abortion is a matter of great public debate "and I can conceive of no reason why advocacy . . . should affect Dr. Koop's fitness to be surgeon general." Said Hatch: "Dr. Koop is a distinguished physician who is eminently qualified to hold this position. Not only is he a renowned surgeon and administrator but throughout his career has engaged in an incredibly numerous and varied set of public health activities."

Dr. Koop, 65, was surgeon-in-chief at Children's Hospital in Philadelphia when he was nominated. During the congressional debate over the nomination, he had been serving in his twin capacity as Deputy Assistant Secretary for Health at the Health and Human Services (HHS) Department.

Hatch said that one objection to Dr. Koop had been that he should not be permitted to oversee or control certain PHS programs. Actually, Dr. Koop must report directly to Edward Brandt, M.D., Assistant Secretary for Health, Hatch noted.

The Senate fight against the nomination was led by Senator Edward Kennedy (D-MA), who criticized what he called "the essentially total absence of training or experience in public health." The Senator also accused Dr. Koop of being "out of touch with the role of women in our society."

Senator John Heinz (R-PA) said Dr. Koop's "personal views on these matters will in no way interfere with the manner in which he carries out the duties of the office of Surgeon General."

Buy U. S. Bonds

Prostatic Carcinoma

(Continued from page 65)

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Pharyngeal Hemorrhage

(Continued from page 54)

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Acoustic Tumors

(Continued from page 68)

should use personal cases for review and comparison and should not merely extrapolate the results achieved by other surgeons when discussing treatment of patients with acoustic tumors. We urge other surgeons who utilize other methods or approaches to report their results.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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Radiation Therapy Procedures

(Continued from page 56)

Dosimetry and Treatment Planning

In addition to simple dosage calculations, hand or computer-produced isodose curves of external beams or intracavitary sources can be performed to obtain a graphic representation of dose distribution of the radiation. The present computer technique provides planar computations, but in the near future, three-dimensional representations will be obtainable.

In addition to the dosage computation, there are other important steps in treatment planning.

- Treatment portals must be simulated in order to relate external landmarks to the patient's internal structures.
- Fluoroscope visualizations and radiographs are necessary for the localization of the tumor volume.
- The tolerance level of normal tissues included in the treated volume must be determined.
- Localization films must be obtained using the therapy unit with the patient in the treatment position. On occasion, verification of treatment plans can be carried out using *in vivo* thermoluminescent dosimeters.
- Immobilization of the patient is critically important in reproducing the treatment volume from day to day. Body casts, head holders, foam pillows, restraining bands, and special vacuum-operated holders are helpful in immobilizing patients.
- Properly designed lead or tungsten shielding blocks are necessary to protect normal uninvolved structures such as eyes, ears, spinal cord, lung, heart, kidneys, liver, and gonads. When possible, individually tailored shielding blocks should be built for each patient.
- Special compensating filters are frequently required to correct the distribution of radiation over an irregular external surface such as thoracic inlet, or with head and neck tumors.

Supportive Care

Patients with side effects from radiation or in less than optimum general condition will need the best supportive care. Acute reactions such as nausea and vomiting must be properly controlled; phenothiazide compounds such as Compazine or Thorazine are usually effective for this condition. If vomiting persists, medication should be given parenterally.

Diarrhea, which frequently occurs with radiation of the abdomen or pelvis, should be controlled with antispasmodics and preparations containing kaolin pectin.

Careful monitoring of fluids and electrolyte balance with periodic laboratory determination is mandatory.

Mucositis of the oral cavity and oropharynx is a frequent complaint after the patient has received a total of 3,000 rads, but it can be significantly diminished by continuous mouth rinsing with lukewarm water or commercial mouth wash preparation mixed with water. Local care of the lips and oral cavity with topical glycerine is helpful. Diet should be adjusted to the swallowing ability of the patient, and soft or liquid food with a high level of protein and carbohydrate may be necessary.

Dental care is important during and after radiation therapy. Special care should be exercised to restore the normal environment of the oral floor and to maintain the best condition of the teeth and gingiva, because the radiation of the salivary glands decreases the production of saliva and changes the chemical composition. Prophylactic fluoride treatment of the teeth following radiation treatment diminishes caries.

Irradiated skin frequently develops changes that vary from erythema to dry or moist desquamation. It is extremely important that the patient's skin be kept clean; it should be washed with mild soap without scrubbing and topical cortisone applied. However, if infection is suspected or present, steroids should not be used; increased use of hydrogen peroxide and topical application of 10-20 per cent zinc oxide are indicated.

Summary

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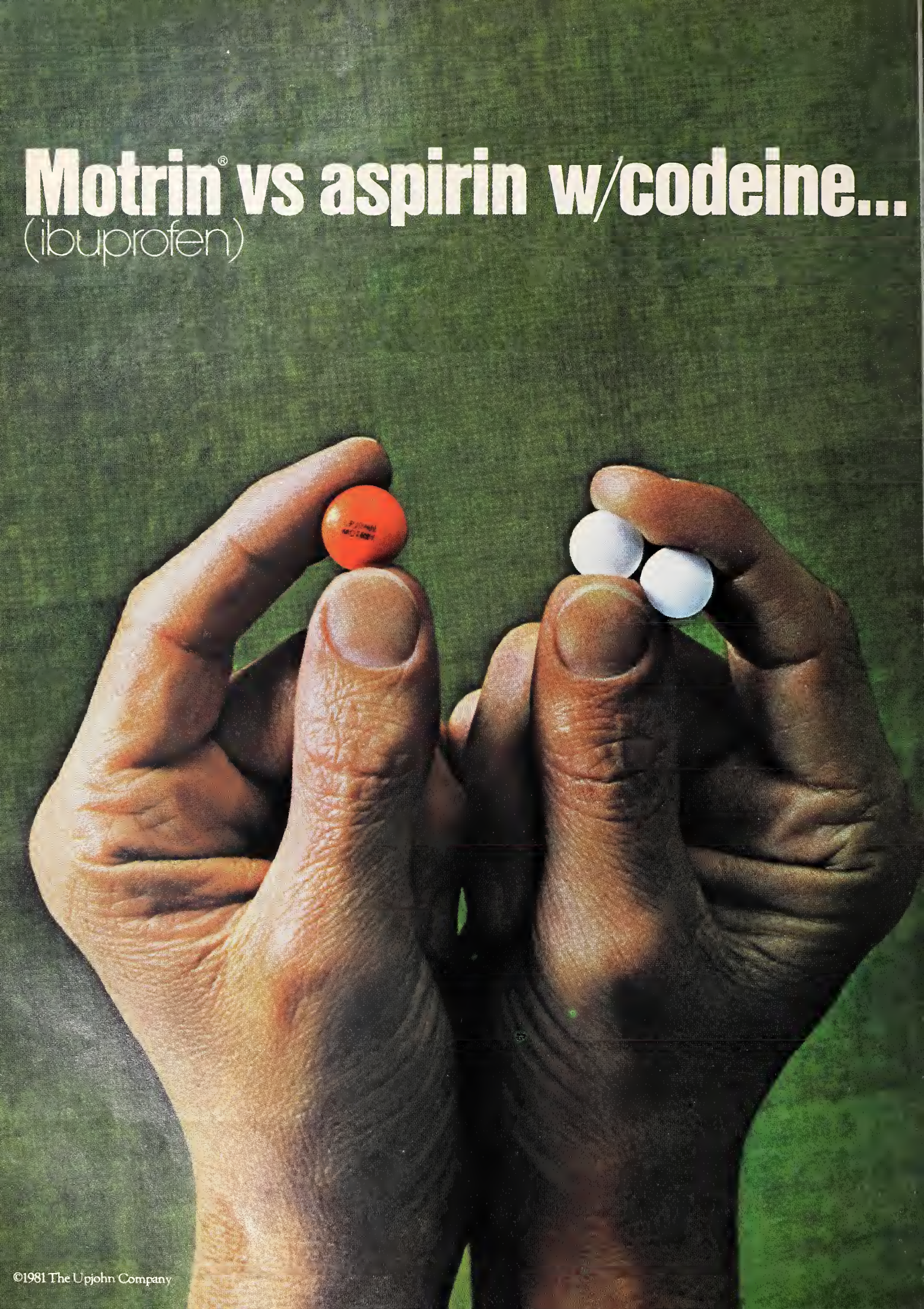


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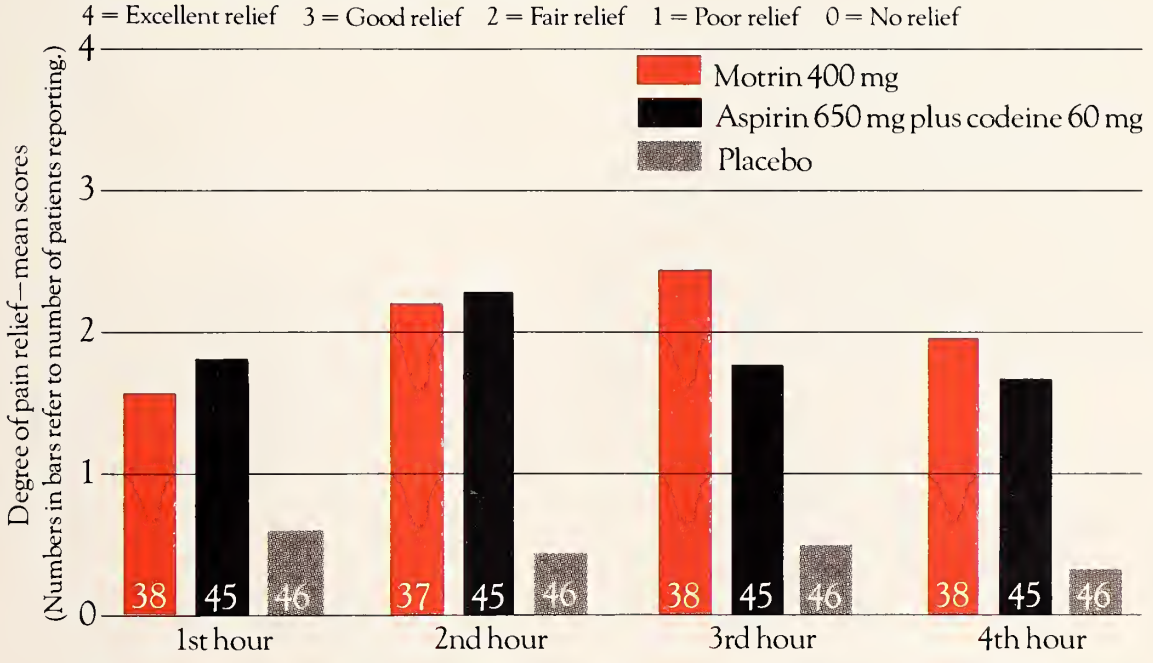


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Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin:* Used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy nor by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea;* epigastric pain;* heartburn;* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness;* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

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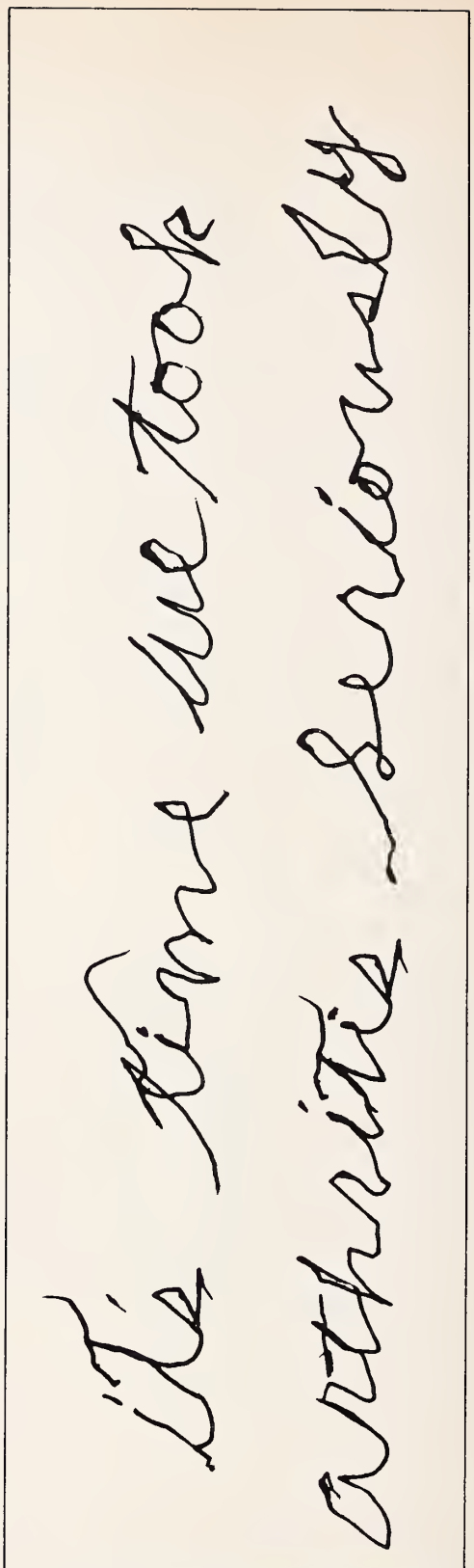
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ARTHRITIS FOUNDATION

AMA House of Delegates

(Continued from page 41)

representatives of the medical community; participation must be structured to maximize the involvement of the medical community. Any peer review process must provide for consideration of the views of individual physicians, groups of physicians, or institutions under review.

5. Peer review evaluations are based on appropriateness, medical necessity, and efficiency of services to assure quality medical care.

6. Any system of medical peer review must have established procedures.

7. Peer review of medical practice and the patterns of medical practice of individual physicians, groups of physicians, and physicians within institutions is an ongoing process of assessment and evaluation.

8. Peer review is an educational process for physicians to assure quality medical services.

9. Any peer review process must protect the confidentiality of medical information obtained and used in conducting peer review.

CME Accreditation

The House of Delegates adopted tighter standards for accrediting continuing medical education. Final approval, however, was withheld until the Delegates have an opportunity to see the handbook to be used to implement these standards. This handbook will be used to evaluate national organizations, medical societies, academic centers, and hospitals that offer CME programs. It is proposed that sponsors of CME courses that advocate unscientific procedures be precluded. In order to receive accreditation, a CME program sponsor must offer instruction in recognized medical subjects that appear in medical school curricula.

Medical Consequences of Nuclear War

The House of Delegates adopted a Board report recommending that the AMA inform the President and Congress of the medical consequences of nuclear war. The action also directed the AMA to educate physicians and the public on the issue. In addition, the AMA will cooperate with other health care organizations and responsible authorities on medical matters in the event of national emergencies. While the Board and the Delegates are concerned about the medical consequences of nuclear war, the action precludes the AMA from involvement in national defense issues outside its professional expertise.

In other actions, the House voted to:

- endorse model legislation to ban the manufacture, sale, and distribution of "look-alike" drugs;
- oppose proficiency testing programs by the Secretary of Health and Human Services to determine the qualifications of clinical laboratory and other health care personnel;
- continue monitoring of federal regulations affecting the practice of nuclear medicine;
- support legislation to delete the three-day prior hospitalization requirement for providing extended care facility benefits under Medicare;
- oppose federal intervention into physicians' prescribing practices;
- encourage further scientific studies on the effects of acid precipitation;
- support adequate funding of biomedical, behavioral, and clinical research activities;
- adopt a policy emphasizing the right of hospitalized patients to choose their own physicians; and to
- request further study of the issue of competition between hospitals and physicians.

We, your Delegates, urge you to let us know your opinions so that we may represent you better. Even if you cannot attend the meeting personally, you can prepare a resolution and request that it be submitted to the House by one of your official Delegates. The next AMA House of Delegates meeting will be in Chicago, June 13-16, 1982.

CLAIR C. CONARD, M.D., *Delegate*
ALEX C. SCOTT, M.D., *Delegate*
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NOMINATING COMMITTEE

The Nominating Committee of the Kansas Medical Society, consisting of Phillip A. Godwin, M.D., Chairman; Drs. L. William Halling, Warren Meyer and John Yulich, submits to the House of Delegates the following list of nominations for the elective offices of the Society. Wherever more than one nomination appears, they are presented in alphabetical order.

President

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Has served as Councilor. Is currently serving as President Elect.

President Elect

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now First Vice President and has chaired the Legislative Committee for many years.

First Vice President

F.Calvin Bigler, M.D., Garden City. Born in 1931. Graduated from Yale University School of Medicine in 1957. Practices General Surgery. Is Past President of the Kansas chapter, American College of Surgeons, and is now serving as Second Vice President of KMS.

Second Vice President

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and Speaker of the KMS House of Delegates.

Jack R. Cooper, M.D., Shawnee Mission. Born in 1917. Graduated from Ohio State University School of Medicine in 1943. Practices Neurosurgery. Is currently serving as Secretary.

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is now serving as AMA Alternate Delegate.

Constitutional Secretary

K. William Bruner, Jr., M.D., Wichita. Born in 1944. Graduated from Harvard Medical School in 1970. Practices Pathology. Is active in Society Committee work.

Virginia T. Gruendel, M.D., Kansas City. Born in 1930. Graduated from the University of Kansas School of Medicine in 1955. Is in Family Practice. Is active in Society Committee work.

Wayne O. Wallace, Jr., M.D., Atchison. Born in 1936. Graduated from the University of Missouri School of Medicine, Columbia, in 1965. Is in Family Practice. Is currently serving as Councilor and is active in Society Committee work.

Treasurer

Richard Meidinger, M.D., Topeka. Born in 1939. Graduated from the University of Kansas School of Medicine in 1965. Practices Diagnostic Roentgenology. Has served as Councilor.

William K. Walker, M.D., Sedan. Born in 1928. Graduated from the University of Kansas School of Medicine in 1945. Is in Family Practice. Is now serving as Treasurer.

Speaker

G. Rex Stone, M.D., Manhattan. Born in 1929. Graduated from the University of Kansas School of Medicine in 1954. Practices General Surgery. Is now Vice Speaker.

Vice Speaker

Franklin G. Bichlmeier, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Cardiovascular Surgery. Is active in Society Committee work.

AMA Delegate

Donald D. Goering, M.D., Coldwater. Born in 1931. Graduated from the University of Kansas School of Medicine in 1956. Is in Family Practice. Is a Past President of KMS and Kansas Academy of Family Practice.

Alex Scott, M.D., Junction City. Born in 1923. Graduated from the University of Wisconsin Medical School in 1948. Is in Family Practice. Is now serving as AMA Delegate.

AMA Alternate Delegate

Warren E. Meyer, M.D., Wichita. Born in 1927. Graduated from the Northwestern University Medical School in 1951. Practices General Surgery. Is a Past President of KMS.

Linda D. Warren, M.D., Hanover. Born in 1944. Graduated from the University of Kansas School of Medicine in 1970. Is in Family Practice. Is active in KMS and AMA Committee work.

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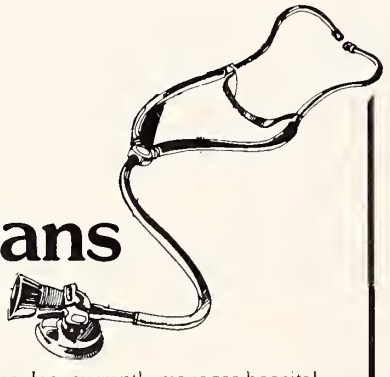
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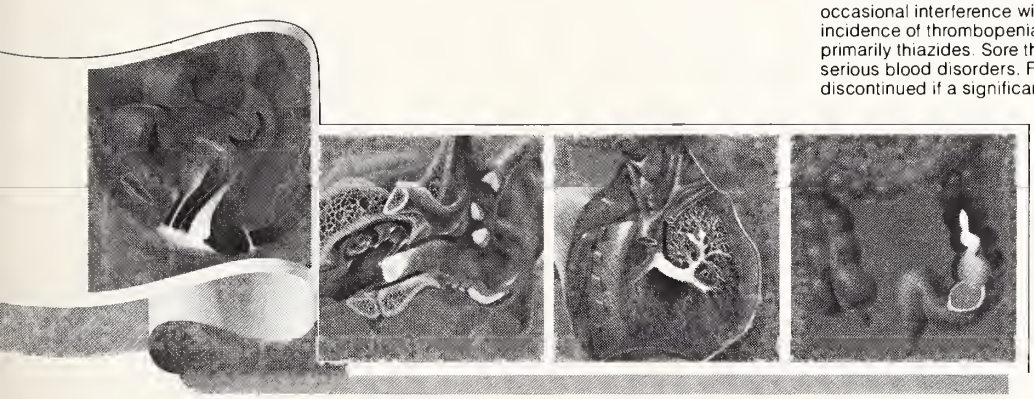
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Expanding its usefulness in antimicrobial therapy



in recurrent UTI...
a continuing record of high clinical effectiveness against common uropathogens

in acute otitis media in children...
effective against both major otic pathogens...with *b.i.d.* convenience

in acute exacerbations of chronic bronchitis in adults...
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in shigellosis...
faster relief of diarrhea than with ampicillin[2]

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. Limited clinical information presently available on effectiveness of treatment of otitis media with Bactrim when infection is due to ampicillin-resistant *Haemophilus influenzae*. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

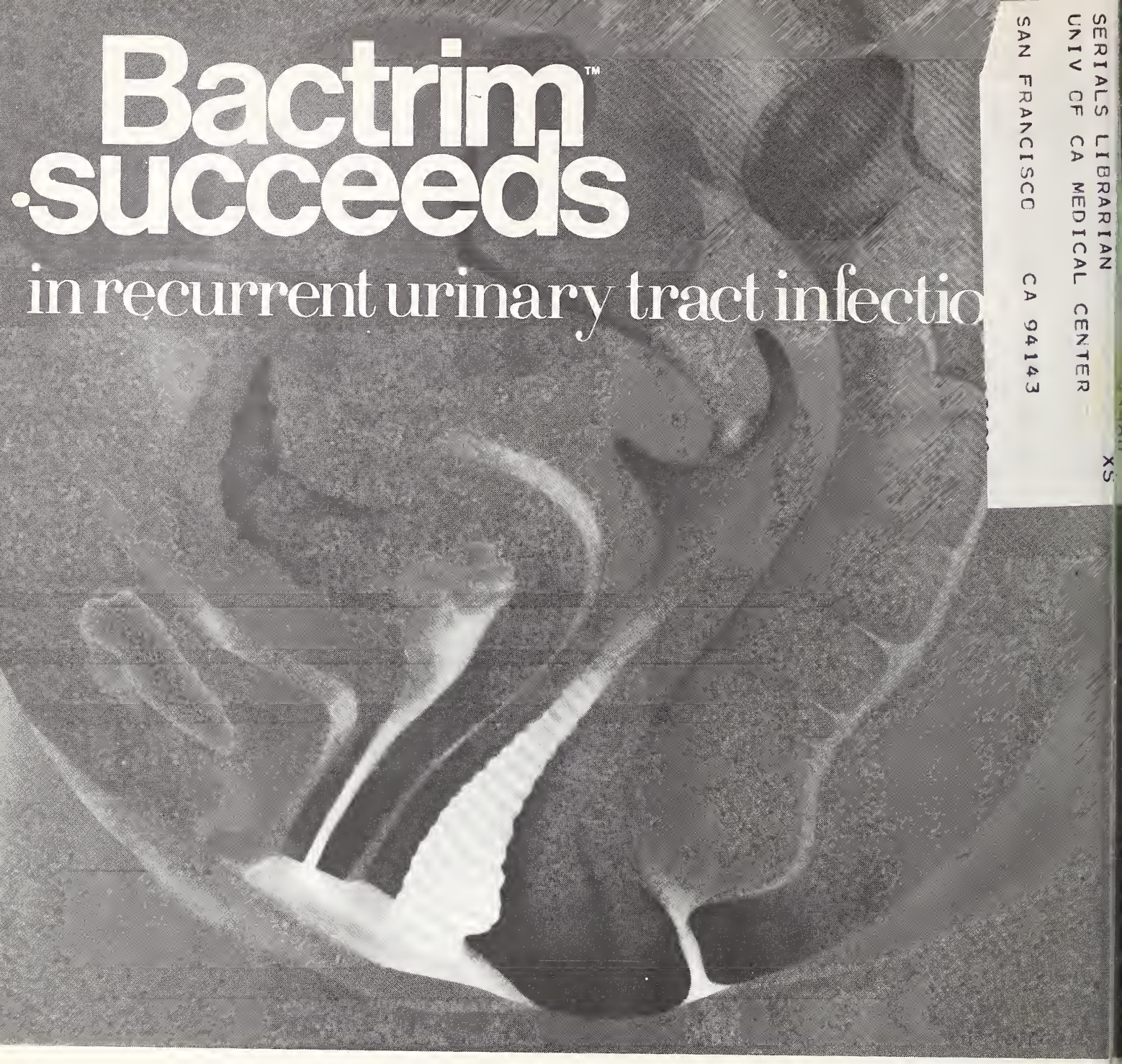
Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry-flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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Bactrim™ succeeds

in recurrent urinary tract infectio



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Bactrim continues to demonstrate high clinical effectiveness in recurrent urinary tract infections. Bactrim reaches effective levels in urine, serum, and renal tissue[1]...the trimethoprim component diffuses into vaginal secretions in bactericidal concentrations[1]... and in the fecal flora, Bactrim effectively suppresses Enterobacteriaceae[1,2] with little resulting emergence of resistant organisms.

1. Rubin RH, Swartz MN: *N Engl J Med 303*:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

Bactrim™ DS
160 mg trimethoprim and 800 mg sulfamethoxazole
DOUBLE STRENGTH TABLETS

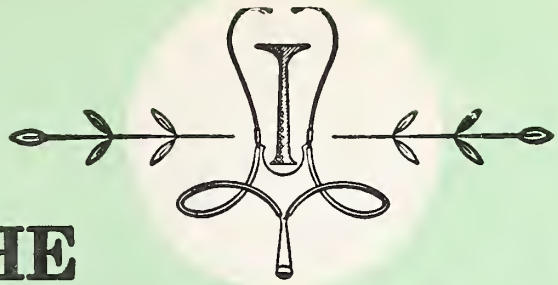
maximizes results with B.I.D. convenience

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*due to susceptible strains of indicated organisms

Please see previous page for summary of product information.

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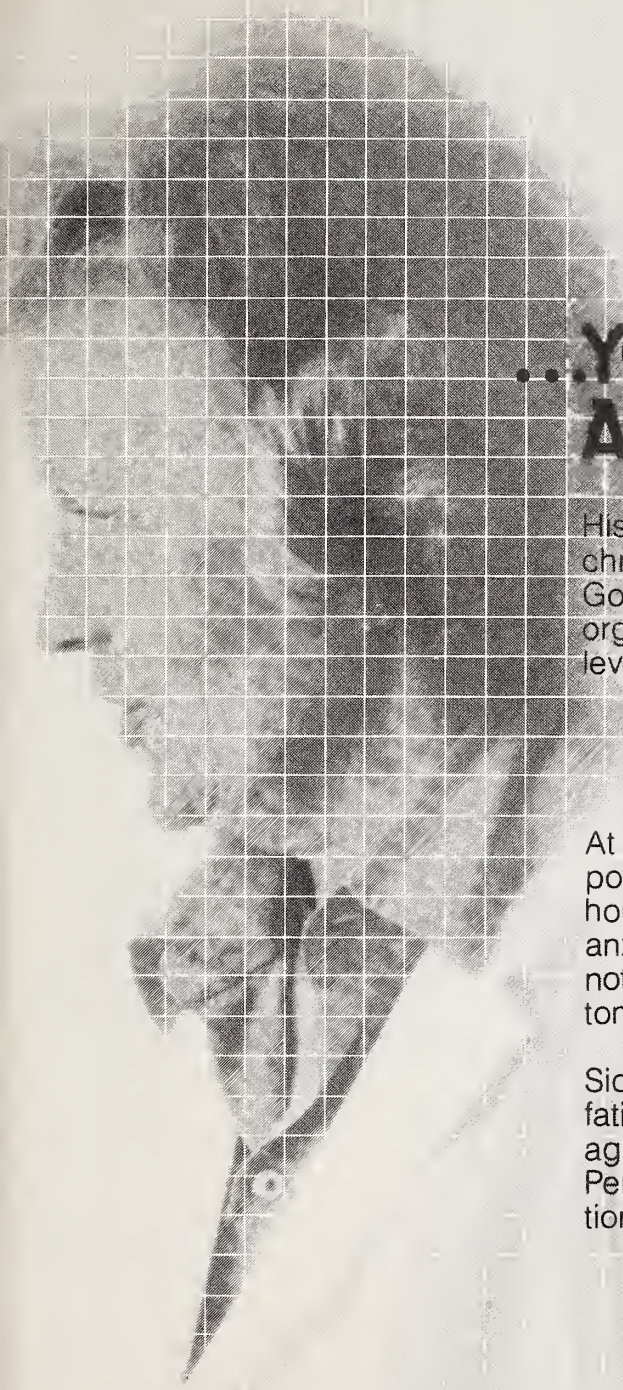
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VOL. LXXXIII
NO. III

THE PATIENT THINKS HE HAS HEART TROUBLE...





...YOU KNOW IT'S REALLY ANXIETY SYMPTOMS

His presenting symptoms: palpitations, chest pain, chronic exhaustion and occasional difficulties in breathing. Good reason for concern. A complete workup uncovers no organic dysfunction, but it *does* reveal excessively high levels of anxiety and apprehension.

For rapid relief you prescribe Valium (diazepam/Roche)

At times like this, Valium (diazepam/Roche) can be a potent therapeutic ally. It works promptly. Within just a few hours, the patient begins to feel calmer. And in a few days, anxiety relief not only becomes more pronounced but a noticeable reduction in anxiety-generated somatic symptoms also occurs.

Equally important, Valium is generally well tolerated. Side reactions more serious than drowsiness, ataxia and fatigue are rare. Patients should, of course, be cautioned against driving or drinking alcohol while on Valium therapy. Periodic reassessment of the need for antianxiety medication should also be performed.

VALIUM® (IV)
diazepam/Roche

2-mg, 5-mg, 10-mg scored tablets

BECAUSE YOU'RE CONVINCED THE PATIENT NEEDS IT

ROCHE®

Please see summary of product information on the following page.

VALIUM®(diazepam/Roche)

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation. The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
How Supplied: For oral administration, Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100* and 500;* Prescription Paks of 50, available in trays of 10.* Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25,† and in boxes containing 10 strips of 10.†

*Supplied by Roche Products Inc., Manati, Puerto Rico 00701
†Supplied by Roche Laboratories, Division of Hoffmann-La Roche Inc., Nutley, New Jersey 07110

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

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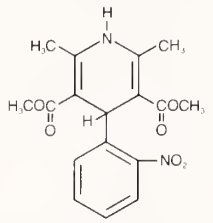
PROCARDIA®
(NIFEDIPINE) *Capsules 10 mg*

Please see PROCARDIA® *prescribing information on next page.*

PROCARDIA® CAPSULES
nifedipine

For Oral Use

DESCRIPTION: PROCARDIA (nifedipine) is an antianginal drug belonging to a new class of pharmacological agents, the calcium channel blockers. Nifedipine is 3,5-pyridinedicarboxylic acid, 1,4-dihydro-2,6-dimethyl-4-(2-nitrophenyl)-, dimethyl ester, $C_{17}H_{18}N_2O_6$, and has the structural formula:



Nifedipine is a yellow crystalline substance, practically insoluble in water but soluble in ethanol. It has a molecular weight of 346.3. PROCARDIA CAPSULES are formulated as soft gelatin capsules for oral administration each containing 10 mg nifedipine.

CLINICAL PHARMACOLOGY: PROCARDIA (nifedipine) is a calcium ion influx inhibitor (slow channel blocker or calcium ion antagonist) and inhibits the transmembrane influx of calcium ions into cardiac muscle and smooth muscle. The contractile processes of cardiac muscle and vascular smooth muscle are dependent upon the movement of extracellular calcium ions into these cells through specific ion channels. PROCARDIA selectively inhibits calcium ion influx across the cell membrane of cardiac muscle and vascular smooth muscle without changing serum calcium concentrations.

Mechanism of Action: The precise means by which this inhibition relieves angina has not been fully determined, but includes at least the following two mechanisms:

1) Relaxation and prevention of coronary artery spasm: PROCARDIA dilates the main coronary arteries and coronary arterioles, both in normal and ischemic regions, and is a potent inhibitor of coronary artery spasm, whether spontaneous or ergonovine-induced. This property increases myocardial oxygen delivery in patients with coronary artery spasm, and is responsible for the effectiveness of PROCARDIA in vasospastic (Prinzmetal's or variant) angina. Whether this effect plays any role in classical angina is not clear, but studies of exercise tolerance have not shown an increase in the maximum exercise rate-pressure product, a widely accepted measure of oxygen utilization. This suggests that, in general, relief of spasm or dilation of coronary arteries is not an important factor in classical angina.

2) Reduction of oxygen utilization: PROCARDIA regularly reduces arterial pressure at rest and at a given level of exercise by dilating peripheral arterioles and reducing the total peripheral resistance (afterload) against which the heart works. This unloading of the heart reduces myocardial energy consumption and oxygen requirements and probably accounts for the effectiveness of PROCARDIA in chronic stable angina.

Pharmacokinetics and Metabolism: PROCARDIA is rapidly and fully absorbed after oral administration. The drug is detectable in serum 10 minutes after oral administration, and peak blood levels occur in approximately 30 minutes. It is highly bound by serum proteins. PROCARDIA is extensively converted to inactive metabolites and approximately 80% of PROCARDIA and metabolites are eliminated via the kidneys. The half-life of nifedipine in plasma is approximately two hours. There is no information on the effects of renal or hepatic impairment on excretion or metabolism of PROCARDIA.

Hemodynamics: Like other slow channel blockers, PROCARDIA exerts a negative inotropic effect on isolated myocardial tissue. This is rarely, if ever, seen in intact animals or man, probably because of reflex responses to its vasodilating effects. In man, PROCARDIA causes decreased peripheral vascular resistance and a fall in systolic and diastolic pressure, usually modest (5–10 mm Hg systolic), but sometimes larger. There is usually a small increase in heart rate, a reflex response to vasodilation. Measurements of cardiac function in patients with normal ventricular function have generally found a small increase in cardiac index without major effects on ejection fraction, left ventricular end diastolic pressure (LVEDP) or volume (LVEDV). In patients with impaired ventricular function, most acute studies have shown some increase in ejection fraction and reduction in left ventricular filling pressure.

Electrophysiologic Effects: Although, like other members of its class, PROCARDIA decreases sinoatrial node function and atrioventricular conduction in isolated myocardial preparations, such effects have not been seen in studies in intact animals or in man. In formal electrophysiologic studies, predominantly in patients with normal conduction systems, PROCARDIA has had no tendency to prolong atrioventricular conduction, prolong sinus node recovery time, or slow sinus rate.

INDICATIONS AND USAGE: I. Vasospastic Angina: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. Chronic Stable Angina (Classical Effort-Associated Angina): PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta-blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Increased Angina/Beta Blocker Withdrawal: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients usually receiving a beta blocker have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event, as the unloading effect of PROCARDIA would be expected to be of less benefit to these patients, owing to their fixed impedance to flow across the aortic valve.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. See Warnings.

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents: See Indications and Warnings. Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates: PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Carcinogenesis, mutagenesis, impairment of fertility: Nifedipine was administered orally to rats for two years and was not shown to be carcinogenic. When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose. *In vivo* mutagenicity studies were negative.

Pregnancy: Pregnancy category C. Nifedipine has been shown to be teratogenic in rats when given in doses 30 times the maximum recommended human dose. Nifedipine was embryotoxic (increased fetal resorptions, decreased fetal weight, increased stunted forms, increased fetal deaths, decreased neonatal survival) in rats, mice and rabbits at doses of from 3 to 10 times the maximum recommended human dose. In pregnant monkeys, doses 2/3 and twice the maximum recommended human dose resulted in small placentas and underdeveloped chorionic villi. In rats, doses three times the maximum human dose and higher caused prolongation of pregnancy. There are no adequate and well-controlled studies in pregnant women. PROCARDIA should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

ADVERSE REACTIONS: In multiple-dose U.S. and foreign-controlled studies in which adverse reactions were reported spontaneously, adverse effects were frequent but generally not serious and rarely required discontinuation of therapy or dosage adjustment. Most were expected consequences of the vasodilator effects of PROCARDIA.

Adverse Effect	PROCARDIA (%) (N = 226)	Placebo (%) (N = 235)
Dizziness, light-headedness, giddiness	27	15
Flushing, heat sensation	25	8
Headache	23	20
Weakness	12	10
Nausea, heartburn	11	8
Muscle cramps, tremor	8	3
Peripheral edema	7	1
Nervousness, mood changes	7	4
Palpitation	7	5
Dyspnea, cough, wheezing	6	3
Nasal congestion, sore throat	6	8

There is also a large uncontrolled experience in over 2100 patients in the United States. Most of the patients had vasospastic or resistant angina pectoris, and about half had concomitant treatment with beta-adrenergic blocking agents. The most common adverse events were the same ones seen in the controlled trials, with dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

Several of these side effects appear to be dose related. Peripheral edema occurred in about one in 25 patients at doses less than 60 mg per day and in about one patient in eight at 120 mg per day or more. Transient hypotension, generally of mild to moderate severity and seldom requiring discontinuation of therapy, occurred in one of 50 patients at less than 60 mg per day and in one of 20 patients at 120 mg per day or more.

In addition, 2% or fewer of patients reported the following: **Respiratory:** Nasal and chest congestion, shortness of breath. **Gastrointestinal:** Diarrhea, constipation, cramps, flatulence. **Musculoskeletal:** Inflammation, joint stiffness, muscle cramps. **CNS**: Shakiness, nervousness, jitteriness, sleep disturbances, blurred vision, difficulties in balance. **Other**: Dermatitis, pruritus, urticaria, fever, sweating, chills, sexual difficulties.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

In a subgroup of over 1000 patients receiving PROCARDIA with concomitant beta blocker therapy, the pattern and incidence of adverse experiences was not different from that of the entire group of PROCARDIA treated patients (see **Precautions**).

In a subgroup of patients with a diagnosis of congestive heart failure as well as angina, dizziness or light-headedness, peripheral edema, headache or flushing each occurred in one in eight patients. Hypotension occurred in about one in 20 patients. Syncope occurred in approximately one patient in 250. Myocardial infarction or symptoms of congestive heart failure each occurred in about one patient in 15. Atrial or ventricular dysrhythmias each occurred in about one patient in 150.

Laboratory tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have already been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

OVERDOSAGE: Although there is no well documented experience with PROCARDIA overdosage, available data suggest that gross overdosage could result in excessive peripheral vasodilation with subsequent marked and probably prolonged systemic hypotension. Clinically significant hypotension due to PROCARDIA overdosage calls for active cardiovascular support including monitoring of cardiac and respiratory function, elevation of extremities, and attention to circulating fluid volume and urine output. A vasoconstrictor (such as norepinephrine) may be helpful in restoring vascular tone and blood pressure, provided that there is no contraindication to its use. Clearance of PROCARDIA would be expected to be prolonged in patients with impaired liver function. Since PROCARDIA is highly protein-bound, dialysis is not likely to be of benefit.

DOSAGE AND ADMINISTRATION: The dosage of PROCARDIA needed to suppress angina and that can be tolerated by the patient must be established by titration. Excessive doses can result in hypotension.

The starting dose is one 10 mg capsule, swallowed whole, 3 times/day. The usual effective dose range is 10–20 mg three times daily. Some patients, especially those with evidence of coronary artery spasm, respond only to higher doses, more frequent administration, or both. In such patients, doses of 20–30 mg three or four times daily may be effective. Doses above 120 mg daily are rarely necessary. More than 180 mg per day is not recommended.

In most cases, PROCARDIA titration should proceed over a 7–14 day period so that the physician can assess the response to each dose level and monitor the blood pressure before proceeding to higher doses.

If symptoms so warrant, titration may proceed more rapidly provided that the patient is assessed frequently. Based on the patient's physical activity level, attack frequency, and sublingual nitroglycerin consumption, the dose of PROCARDIA may be increased from 10 mg t.i.d. to 20 mg t.i.d. and then to 30 mg t.i.d. over a three-day period.

In hospitalized patients under close observation, the dose may be increased in 10 mg increments over four to six-hour periods as required to control pain and arrhythmias due to ischemia. A single dose should rarely exceed 30 mg.

No "rebound effect" has been observed upon discontinuation of PROCARDIA. However, if discontinuation of PROCARDIA is necessary, sound clinical practice suggests that the dosage should be decreased gradually with close physician supervision.

Co-Administration with Other Antianginal Drugs: Sublingual nitroglycerin may be taken as required for the control of acute manifestations of angina, particularly during PROCARDIA titration. See **Precautions, Drug Interactions** for information on co-administration of PROCARDIA with beta blockers or long-acting nitrates.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA Capsule contains 10 mg of nifedipine. PROCARDIA Capsules are supplied in amber glass bottles of 100 capsules (NDC 0069-2600-66).

The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77°F (15° to 25°C) in the manufacturer's original container.

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KaMPAC MEMBERS 1982

The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

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Regular	60—R

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LET'S TALK BUSINESS

A continuing series on business communication issues.

Managing Information Costs in the 80's

Spectacular advances in technology are here today, adding new tools to the manager's arsenal at an incredible rate. Especially in the telecommunications area.

To be an effective manager, it's vital that you stay informed about these sophisticated new tools, and how they can help increase your company's productivity by controlling communication costs.

Let's examine some of them, and how they might affect your business.

Do it yourself communications.

There was a time when business growth and change equalled communication expense and confusion. Today's flexible communication systems allow you to change station numbers and features to match changing personnel and business conditions. You have the option to do it yourself, when you need to, and as often as you need to, without the expense of a service call.

The advantage of reduced installation charges through internal programming can be tremendous.

System Control.

As managers' dependence on long distance to effectively run their businesses increases in the 80's, new systems to control those costs make dollars and sense.

For instance, there's more than one way to route a long distance call. One system can automatically search out the least expensive route, and lower overall long distance charges in the process.

Control of long distance calling is also important. You may want to limit certain stations to local calls only, while others have access to toll lines. Some systems can even limit calls only to the area codes you select.

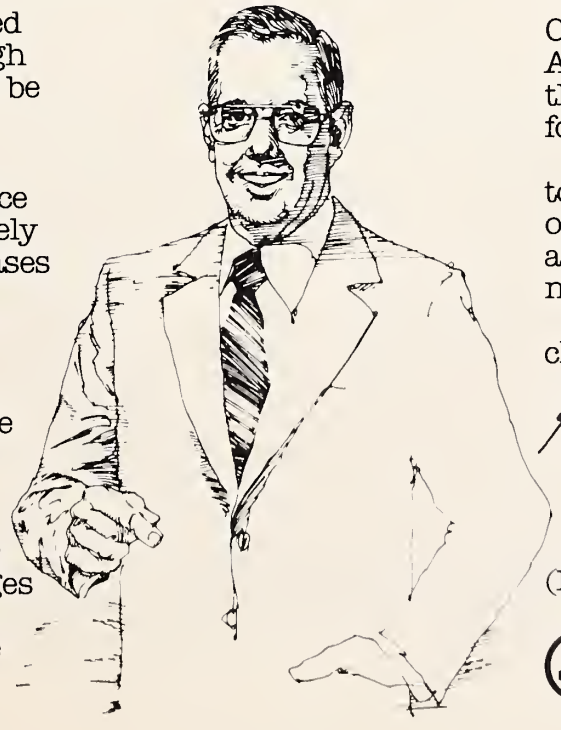
Even more sophisticated cost management systems can track telephone expenses by department, project and client, providing invaluable information to the manager interested in keeping a handle on communication costs.

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For example, one system has the capability of accumulating data by day for batch transmission at night, when rates are lower. The payoff in cost-reduction can be substantial.

Advanced capabilities such as this are already helping businesses to more cost-effectively manage their communication systems.



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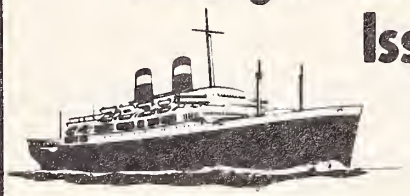
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it's shorter acting, with less accumulation*

In contrast to long-acting benzodiazepines, Ativan has a short, 12-hour half-life, and no active metabolites. In multiple-dose therapy, Ativan accumulates for only two to three days before reaching steady state; the long-acting benzodiazepines—diazepam CIV, chlordiazepoxide CIV, clorazepate CIV and flurazepam CIV—with their active metabolites—accumulate for as long as 20 days, increasing the likelihood of excessive sedation.

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Most benzodiazepines undergo oxidative metabolism and thus utilize the hepatic microsomal enzyme system. Ativan® (lorazepam), however, is metabolized by glucuronidation and does not compete with other drugs for cytochrome P450. Thus, when Ativan is given with Tagamet® (cimetidine), for example, clearance is not delayed, nor sedation increased†—unlike reported observations with patients on other benzodiazepines.[1-5]

1. Jotz U, Reimann I N Engl J Med *302* 1012-1014, 1980
2. Desmond PV, Patwardhan RV, Schenker S, et al Ann Intern Med *93* 266-268, 1980
3. Patwardhan RV, Yarborough GW, Desmond PV, et al Gastroenterology *79* 912-916, 1980
4. Sellers EM, Naranjo CA, Peachey JE N Engl J Med *305* 1255-1262, 1981.
5. Ruffalo RL, Thompson JF, Segal JL South Med J *74* 1075-1078, 1981

*Pharmacokinetics cannot as yet be directly related to efficacy

†All benzodiazepines produce additive effects when given with CNS depressants such as barbiturates or alcohol

it gives you greater control of therapy

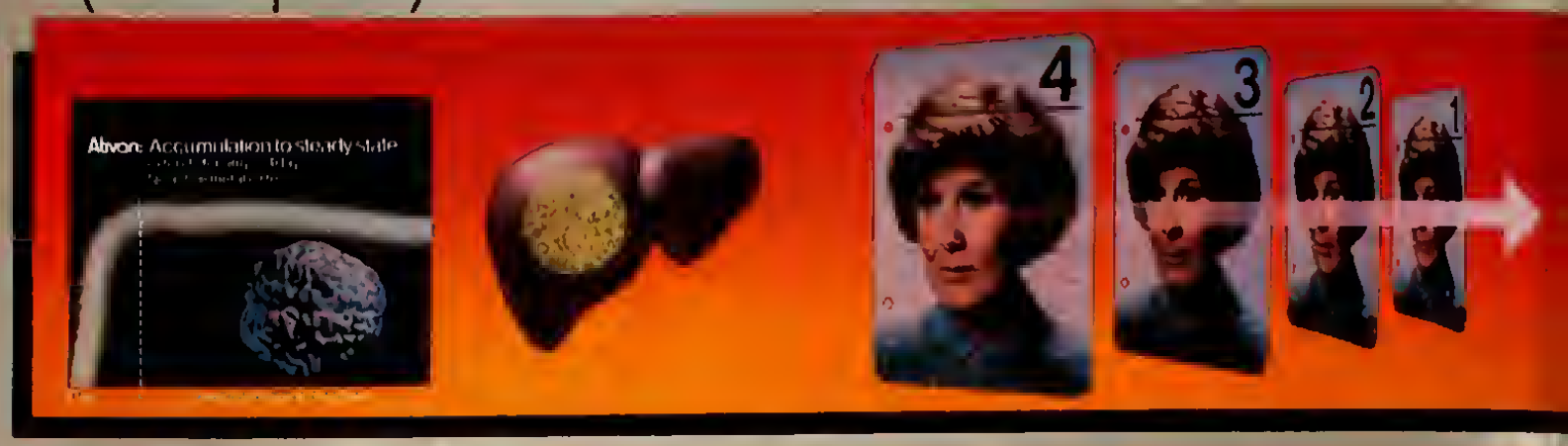
The short half-life of Ativan® facilitates more rapid response to dosage adjustments, allowing you to titrate therapy to patients' changing needs. Also, once you decide to discontinue Ativan, it will be out of your patient's system four days after the final dose—in contrast to long-acting benzodiazepines and their active metabolites which take as long as two weeks to be totally eliminated.

Ativan®
for (lorazepam) CIV
Anxiety

See important information on following page



Ativan®: Agent of Change
(lorazepam) ℞



- Little accumulation lessens likelihood of excessive sedation
- Unlike most benzodiazepines, Ativan does not compete with other drugs, such as Tagamet® (cimetidine), for the microsomal enzyme system during biotransformation
- Metabolism not affected by age or liver dysfunction
- Short half-life provides greater control of therapy
- Promptly eliminated from patient's system after discontinuation
- Specifically evaluated and found effective for anxiety associated with cardiovascular and gastrointestinal disorders
- A distinctive change from long-acting benzodiazepines, all of which have active metabolites and are much the same

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence. Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating) Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day) Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G I disease Safety and effectiveness in children under 12 years have not been established

ESSENTIAL LABORATORY TESTS Some patients have developed leukopenia, some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy

CLINICALLY SIGNIFICANT DRUG INTERACTIONS Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS No evidence of carcinogenic potential emerged in rats during an 18-month study No studies regarding mutagenesis have been performed

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses Clinical significance of these findings is not known However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%) Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P Usefulness of dialysis has not been determined

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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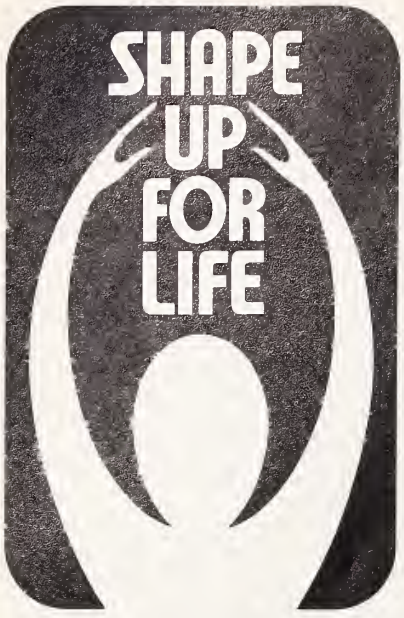
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

The radio is predicting yet another snowstorm and confirming that the winter of '82 is the worst this century, while I try to conjure up the vision of a beautiful spring day and the beginning of our convention.

It was again my privilege to meet with the Kansas Council of Women at their annual meeting in Topeka. This council was organized in 1911 to unite the women of Kansas for action on issues designed to promote the public welfare. Our own Katie Pyle (Lucien R.), Topeka, is chairman of the Legislative Committee and is actively involved in rules, regulations, and resolutions pertaining to nursing homes. My speech at the evening banquet related to the nature of our organization, its concerns, and the major committees we work with.

At this writing, I look forward to our meeting scheduled in Topeka on March 2-3. We hope to balance the budget, tie up loose ends of the past year's business, and meet with our legislators — the primary reason for the meeting. It is always a pleasure to visit the Statehouse and to observe the process of law-making — and perhaps be a small part of it.

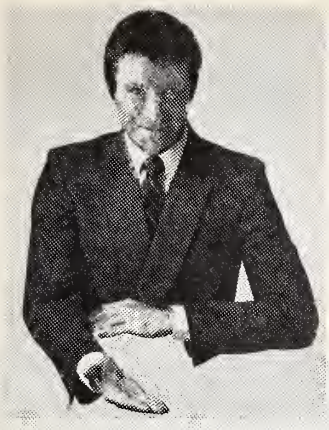
Convention time is near, and we hope for a good attendance. Wichita is always a fun place, and under the capable direction of Georgann Richardson (S. F.) and her hard-working crew, it's sure to be a great meeting. Our representative from the National Auxiliary this year will be Isobel Dvorsky (Harry S.), President. She will bring you brief greetings during the opening House of Delegates session. Our agenda will also include AMA Trustee Robert T. Kelly, M.D., and John-Henry Pfifferling, Ph.D., from the Center for Well Being of Health Professionals in North Carolina.

My year of new challenges, goals, and opportunities is drawing to a close. I have attempted to share our Auxiliary activities in a brief, concise manner with the hope that you have found it interesting. This has been a most rewarding year, and it is with mixed emotions that I bid you farewell. I have enjoyed it immensely — but will not miss the many deadlines.

In early May, I will pass the gavel of leadership to Meldon Laury (David G.); under her expert guidance, the many ongoing programs supporting and promoting your profession will continue.

Thank you!

Betty L. Moore,
President
Kansas Medical Society Auxiliary



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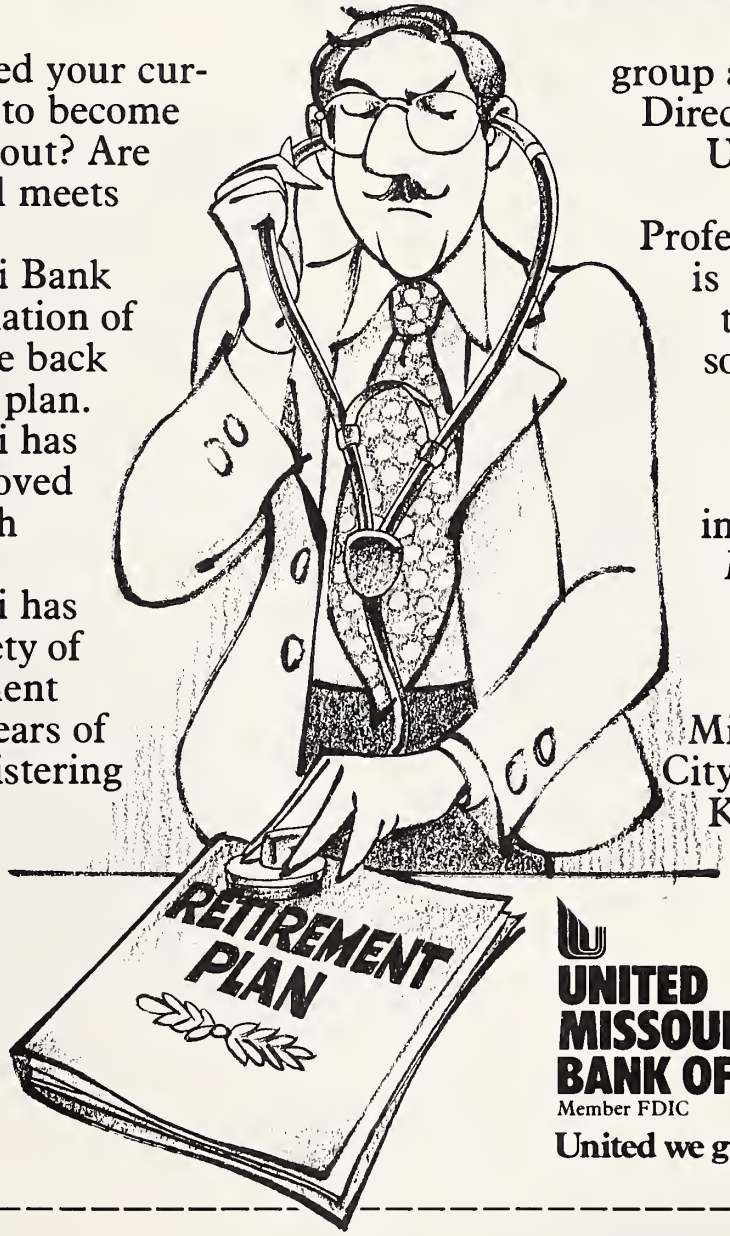
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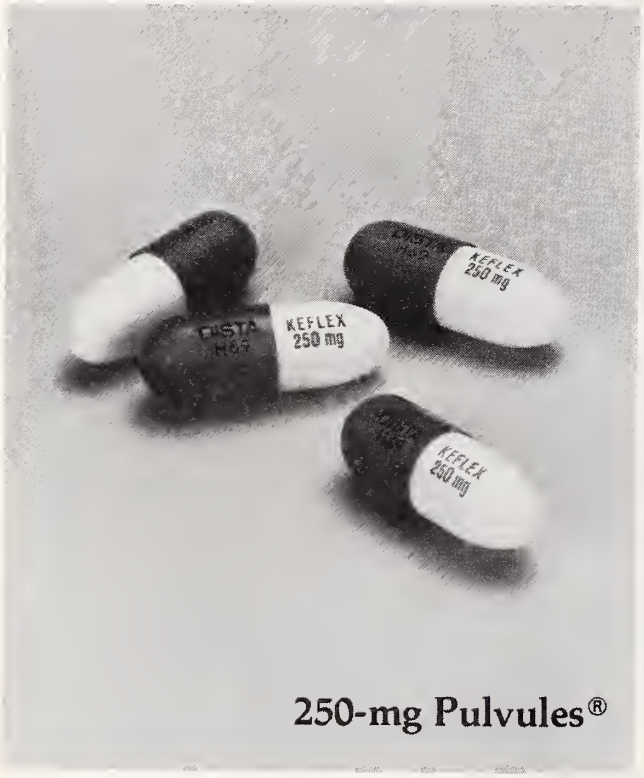
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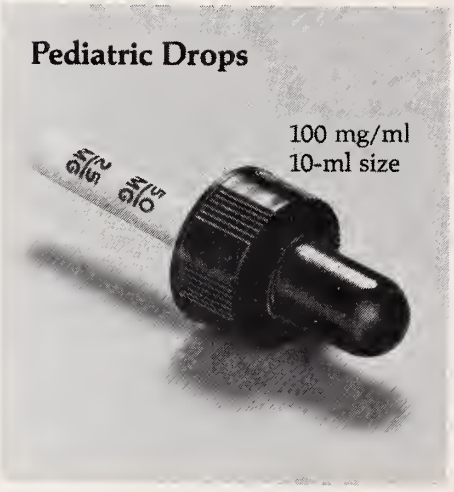
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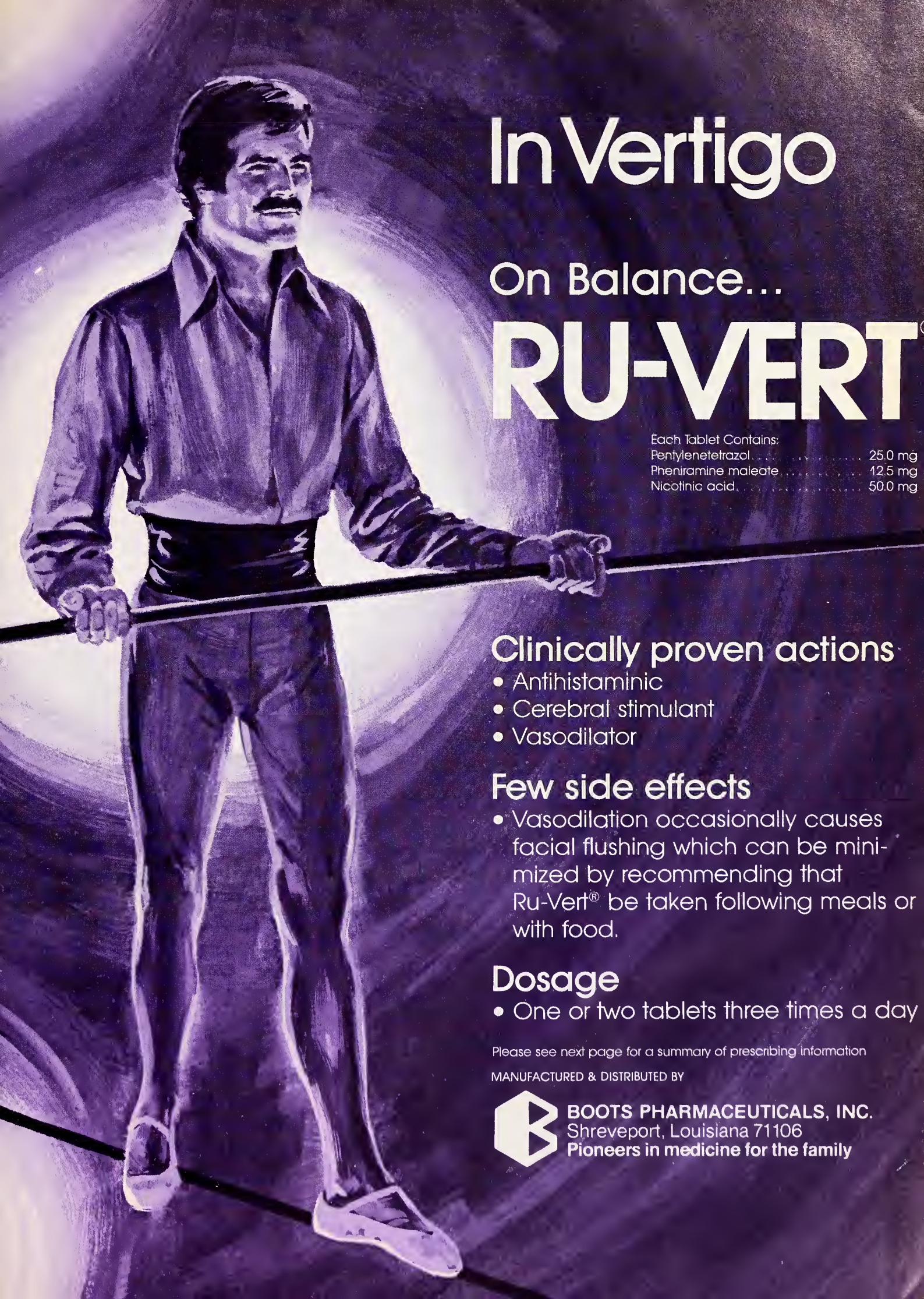


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NOMINATING COMMITTEE

The Nominating Committee of the Kansas Medical Society, consisting of Phillip A. Godwin, M.D., Chairman; Drs. L. William Halling, Warren Meyer and John Yulich, submits to the House of Delegates the following list of nominations for the elective offices of the Society. Wherever more than one nomination appears, they are presented in alphabetical order.

President

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Has served as Councilor. Is currently serving as President Elect.

President Elect

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now First Vice President and has chaired the Legislative Committee for many years.

First Vice President

F. Calvin Bigler, M.D., Garden City. Born in 1931. Graduated from Yale University School of Medicine in 1957. Practices General Surgery. Is Past President of the Kansas chapter, American College of Surgeons, and is now serving as Second Vice President of KMS.

Second Vice President

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and Speaker of the KMS House of Delegates.

Jack R. Cooper, M.D., Shawnee Mission. Born in 1917. Graduated from Ohio State University School of Medicine in 1943. Practices Neurosurgery. Is currently serving as Secretary.

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is former chairman of the Commission on education, and is now serving as AMA Alternate Delegate.

Constitutional Secretary

K. William Bruner, Jr., M.D., Wichita. Born in 1944. Graduated from Harvard Medical School in 1970. Practices Pathology. Is active in Society Committee work.

Virginia T. Gruendel, M.D., Kansas City. Born in 1930. Graduated from the University of Kansas School of Medicine in 1955. Is in Family Practice. Is active in Society Committee work.

Wayne O. Wallace, Jr., M.D., Atchison. Born in 1936. Graduated from the University of Missouri School of Medicine, Columbia, in 1965. Is in Family Practice. Is currently serving as Councilor and as Chairman of the Drug Utilization Review Committee.

Treasurer

Richard Meidinger, M.D., Topeka. Born in 1939. Graduated from the University of Kansas School of Medicine in

(Continued on page 124)

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Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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Thirty-Sixth Annual University of Kansas School of Medicine Issue

The Dean's Letter — 1982

The University of Kansas School of Medicine-Kansas City

MARVIN DUNN, M.D.* *Kansas City, Kansas*

I AM PLEASED to address the readers of the JOURNAL OF THE KANSAS MEDICAL SOCIETY. I have just completed my first year as Dean of the University of Kansas School of Medicine, and would like to bring to your attention some Medical School activities and some of our accomplishments.

Enrollment in the School of Medicine is now 800 students — 200 in each class. One hundred of these future physicians are completing their clinical clerkships at UKSM-Wichita. Renovation of the E. B. Allen Hospital, the permanent home of the Wichita campus, is scheduled for completion in 1982.

The new Bell Memorial Hospital has now been operational for nearly two years. This well-planned facility provides excellent medical care, as well as training physicians and allied health personnel. The Mid-America Radiation Center is fully operational, and its clinical and research activities are increasing annually. The new parking facility provides easy access to Bell Memorial Hospital for patients and their families.

The Division of Health Care Outreach has been increasing its involvement in community based educational programs for health professionals. This includes visits by students to community physicians, locum tenens, placement of off-year medical graduates, assistance to communities recruiting physicians, and consultation services to community physicians and hospitals. In the coming year we will place the first small group of physicians who were Kansas Medical Scholarship recipients and are now ready to enter practice. Our faculty, residents, and students have participated in more off-campus programs than in any previous year in Medical School history. The medical faculty, in conjunction with physicians from participating communities, are conducting problem oriented continuing education programs in a number of Kansas communities.

During the past year, a number of faculty changes have resulted in improved and expanded services. Research and development programs are underway in the areas of recombinant DNA technology, cardiac metabolism and sports medicine, epithelial transport mechanisms, vitreous surgery, and corneal and anterior segment disease. Additional faculty have been added in various departments.

We have received a $50,000 gift from the Sterling Drug Company. Interest from this money will endow an annual W. Clarke Wescoe Lectureship in Pharmacology. A chair in cancer research has also been endowed, and a search committee is currently selecting the recipient. A new appointment will also be made to the currently vacant Hashinger Chair of Gerontology.

The Todd L. Sutherland Center for Pediatric Deformity Microsurgical Center has been dedicated and is now functional. Development of this center has facilitated the new and unique program of maxillo cranial surgery which will permit children with congenital cranio-facial malformations, patients with facial derangements from cancer, and patients with traumatic facial deformities to obtain corrective surgery utilizing these rapid modern techniques.

The Trauma Center is operating successfully, and several seriously injured patients from the Hyatt Regency disaster were cared for here. The Thoracic and Cardiovascular Surgery Program has been expanded, and more than 250 open heart operations were performed last year.

The projected completion date for the new Health Sciences Library is May 1983. This expanded facility is sorely needed.

We have many old and new programs designed to serve the people of Kansas, and we strive constantly to bring them the newest technology and advances in health care. It is our goal to develop educational, research, and clinical programs to assure the highest quality of training to our students and house officers.

We are indebted to you who have supported us, and we hope that our efforts will assure confidence in the medical school and its programs so that we can continue to merit your support.

* Dean, University of Kansas School of Medicine-Kansas City

First Honors Convocation Address

Not To Woo Honor, But To Wed It

ROBERT P. HUDSON, M.D., *Olathe*

IT IS A MELANCHOLY fact of life that most of the recognitions accorded us during our earthly sojourn are more apparent than real. Occasions that appear to honor us are, in reality, for insignificant accomplishments, or in other instances the ceremony is directed at others altogether.

Consider for a moment the more important occasions on which we are recognized in one ceremonial fashion or another. Birth, as a logical first example, despite having been called by one pessimist the "first and direst of all disasters," is celebrated by a variety of rituals ranging from the ringing of bells to the snipping of foreskin. There is no discounting the importance of birth, an event without which we would all be off to a poor start indeed. But the recognition at parturition, as far as the principal character is concerned, is no recognition at all. It lacks a *sine qua non* of meaningful recognition, which is awareness that one is being honored. The recognition of birth clearly is more for the "birthers" than the "birthee." There are then annual reminders of our birthday which, however, if stripped of all accoutrements, mark no particular accomplishment beyond survival which, admittedly in some cases, is accomplishment enough to deserve recognition.

Death, while perhaps not a next logical consideration, must be mentioned here because it suffers the same deficiency as birth. Romeo, for example, must have received precious little gratification from the immortal recognition accorded him by Juliet's suicide. In the same literary vein, but from a somewhat different point of view, few of us are permitted the delicious recognition savored by Tom Sawyer as he surreptitiously audited his own funeral. For most of us, the tragic truth of posthumous acknowledgement is that we are recognized for merely accomplishing death, a curious recognition indeed, since dying is carried off with admirable aplomb by ants, squids, dachshunds, and a host of other ordinarily unrecognized creatures.

There is an hour or two focused on us when we marry, but this is recognition open to all, and attained by most. In general it requires less in the way of talent than tenacity, and thus is not recognition so much of accomplishment as of social conformity — indeed, perhaps even of genetic determinism.

For an unfortunate few there will be a gold watch at retirement, which may represent nothing more than a quest for atonement on the part of the superiors who treated them so shabbily during 35 years of faithful employment. In any event, does the new retiree, with nothing but time on his hands, really need to square his remaining minutes and seconds with the Naval Observatory at Bethesda?

Along the way, for most of us, there will be other honors — a blue ribbon for a foot race, a merit badge for a cigar box full of beetles, a certificate attesting to 12 months of unflagging attendance at Sunday School. And these are important at their moment, but later are perceived to confer roughly the same distinction as learning to dance disco or winning the door prize.

The exception to all this, the one important exception, is the recognition that reflects genuine achievement. It is in the attainment of excellence in worthwhile pursuits that human beings truly can distinguish themselves in a meaningful way. Unfortunately, in terms of genuine merit, such recognition is difficult to come by in the United States. It is the nature of democracy to extol the average and to shun all suggestion of elitism, whether natural or earned. This inborn error of political metabolism was embedded in our national consciousness by the biologically ridiculous conviction put forth in the Declaration of Independence which held "that all men are created equal. . . ." It would have been more in keeping with natural reality had we adopted the phraseology of the earlier Virginia Declaration of Rights which stated "That all men are by nature equally *free* and *independent*. . . ."

This particular weakness of egalitarianism was perceived 150 years ago by the young French nobleman, Alexis de Tocqueville, who arrived in the United States in 1831 to try to decide if the new

* Delivered by Dr. Hudson on October 1, 1980, at the University of Kansas School of Medicine-Kansas City.

Address reprint requests to Dr. Hudson, 12925 Frontier Road, Olathe KS 66061.

democracy of America might serve to supplant the outworn aristocratic regimes of Europe. De Tocqueville wrote, "I confess that I apprehend much less for democratic society from the boldness than from the mediocrity of desires. What appears to me most to be dreaded is, that in the midst of the small incessant occupations of private life, ambition should lose its vigor and its greatness; that the passions of man should abate, but at the same time be lowered; so that the march of society should every day become more tranquil and less aspiring."[1]

Ambrose Bierce put it more strongly in his typical acerbic fashion when in the Devil's Dictionary he defined "equal" as an adjective to mean "As bad as something else," and "equality" the noun in this way, "In politics, an imaginary condition in which skulls are counted instead of brains, and merit is determined by lot and punished by preferment."[2]

In this instance we have evolved much as De Tocqueville predicted we might. Our system of recognition has been pegged firmly to the dollar, and within that system we reward our athletes more than our scientists, our rock stars more than our humanists, and our electricians more than our teachers. Our Congressmen represent the only elite group in our nation that is free to assess its own worth and reward itself accordingly, and perhaps this is an argument against granting the elite any such power.

None of this is meant to denigrate affirmative action programs, or Head Start, or equal pay for equal jobs for women, or any other social device designed to give us all a reasonable chance of getting to the starting line with our potential at that point fully developed. But once the race begins we must recognize that some will run farther and faster than others, and that they should be encouraged to do so, and rewarded with proper recognition when they reach a given level of excellence.

The privileged relationship between student and teacher is almost as old as the history of medicine itself. In the Oath associated with the Hippocratic school of medicine we find the admonition that the student shall "reckon him who taught me this Art equally dear to me as my parents, to share my substance with him and relieve his necessities if required. . . ." In such specifics as sharing substance, medical teachers, probably wisely, have not attempted to redeem the Oath in recent years. Indeed, in our time, there may be instances in which teachers would not care to be reckoned equally with a student's parents. But the importance of the passage does not lie in its literal interpretation. The skeletal significance is simply that students incur a debt of gratitude to their medical teachers, and that the more dedicated and competent the teacher, the greater the debt. For their part, throughout history, the better teachers have perceived their calling, with all its attendant responsibilities, as a human endeavor of the highest order.

Sir William Osler, who came to represent the ideal physician-teacher-humanist at the turn of the 20th century once said, "I desire no other epitaph — no hurry about it, I may say — than the statement that I taught medical students . . . I regard this as by far the most useful and important work I have been called on to do."[3]

It would be fatuous to pretend that all medical teachers have shared Sir William's lofty sentiment. Some simply have no gift for teaching, and do not care enough to repair even their remediable deficiencies. These are the ones who led W. H. Auden to define a professor as "one who talks in someone else's sleep." Others look upon their teaching assignments as evils to be tolerated only because they make possible the basic or clinical research that brings the national and peer accolades that teaching never can. Again from Osler, "In the hurly-burly of today, when the competition is so keen and there are so many seeking the bubble reputation at the eyepiece and the test tube, it is well for young men to remember that no bubble is so irridescent or floats longer than that blown by the successful teacher."[4]

It is difficult for the dedicated teacher to keep Osler's sentiment in mind when, at promotion time, teaching prowess is repeatedly discounted in favor of research and writing. But the best of them somehow persist in their belief in the value of good teaching. It is even possible that, constitutionally, they cannot do otherwise. Perhaps we are indebted to the poor and unmotivated teachers among us for making us appreciate excellence in teaching all the more, and for giving meaning to convocations such as this.

As for our students, we have come a long way from those days of the mid-19th century when Daniel Gilman, President of Johns Hopkins University, could say with considerable justification, that "The medical student is likely to be one son of the family too weak to labour on the farm, too indolent to do any exercise, too stupid for the bar and too immoral for the pulpit."[5]

Today our students quite literally are the *créme de la créme,* at least intellectually, although I'm not certain they are necessarily any better qualified for the pulpit than their 19th century counterparts. In a

(Continued on page 156)

The Peter T. Bohan Lecture, 1981

Scholars, Saints, Skeletons — A Candid Recollection of Professors and What I Learned From Them

STANLEY R. FRIESEN, M.D., Ph.D., ***Kansas City, Kansas***

IT IS WITH REAL HUMILITY and grateful thanks that I add yet another Bohan Lecture to the impressive list of prestigious lectures in the past. There are alumni, friends and students who, together with me, savor their happy memories from medical school and I hope to kindle the loving thoughts all of us have for our professors in the past. I, myself, have had the unique opportunity of being both a medical student (about 40 years ago) and a member of the faculty (beginning 32 years ago) in our beloved institution, the University of Kansas School of Medicine. Although I recall very little of the patients I saw as a student, I do recall my professors vividly. Of them I would like to revive some personal recollections and what I have learned from them.

It is fitting that we begin with Peter T. Bohan, for whom this lectureship is named. He has become virtually a saint around UKSM and was one of the first Professors of Clinical Medicine whom I remember. He was one of the best teachers of medicine. After graduating from Rush Medical College in 1900 and taking postgraduate work in London, Edinburgh, Berlin and Vienna, he came to Kansas City in 1907. The first record of his teaching at UKSM is in 1908. A delightful brochure entitled, "The Man Who Became a Tradition," compiled and written by Helen Sims of our alumni association, reveals his influence on the students of this school. His sword-like sharp sayings by now are legend. Among them are, "He may be a hell of a professor, but he'd starve to death in a county seat town." Peter Bohan would make sure his patients understood his instructions: "What we mean by 'going to bed' is to take off your clothes, put on a nightgown, and stay in bed except to go to the bathroom."

Another Bohanism, "Only neurotics complain of coated tongues." So I've learned a very basic thing from Peter Bohan, "Examine the patient thoroughly, the patient may be a neurotic."

Delivered by Dr. Friesen, Alumnus of the Year, at the annual University of Kansas Medical Alumni banquet on May 15, 1981.

Address reprint requests to Dr. Friesen, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

No history of the Medical Center would be complete without genuflection to Simeon Bishop Bell. His initial gift of 7½ acres and $45,000 resulted in the naming of three different hospitals for him and his wife: the original Eleanor Taylor Bell Memorial Hospital on Goat Hill, built during the first part of this century; the 1924 Bell Memorial Hospital on Rainbow Boulevard at 39th Street; and our present one, completed in 1979.

There were others of importance in our early history. Dr. Don Carlos Guffy was the first Professor of Obstetrics and Gynecology. He was instrumental in raising the money for the 1924 Bell Memorial Hospital. Dr. Frank C. Neff was the first Professor of Pediatrics, and he was influential in raising the money for the children's wing of that Hospital. Dr. Richard L. Sutton was our first Professor of Dermatology and was remembered for that as well as his wild game hunting. His son, Richard Sutton, Jr., also became Professor of Dermatology, succeeding Dr. Charles C. Dennie. Dr. Sam Roberts, Professor of Otorhinolaryngology, came to this medical school in 1914, the same year as did Dr. Major. Dr. E. J. Curran was the first Professor of Ophthalmology. Many of us will recall Dr. Olaf Stoland, Professor of Physiology, for whom a student lounge is named. Dr. Noble P. Sherwood was our venerated Professor of Bacteriology and was the father of Mrs. David W. Robinson. Many of us also recall Dr. James Weaver, Professor of Orthopedics, and Dr. Larry Woods, Professor of Pulmonary Medicine.

One of the most important men in the development of this medical school was Dr. Mervin Sudler, a surgeon, and the first Dean of Scientific Medicine. He came here from Johns Hopkins University. Dr. Major relates in his historical account that when Dr. Sudler came from the East he was warned about the possibility of wild and marauding Indians in Kansas. Can you imagine his anxiety when he stepped off the train at the Kansas City depot and saw real Indians there all painted and feathered? It just happened that Buffalo Bill and his Wild West Show — complete with a troop of Indians — were changing trains on tour through the United States.

Dr. Walter S. Sutton, an early surgeon in our school, gained fame as a geneticist. His work, which was carried out in grasshoppers at the University of Kansas before medical school, resulted in the Sutton-Bovary Hypothesis which states that chromosomes are the repository of genes (1903). Unfortunately, his life was terminated early because of complications of appendicitis.

Dr. William W. Duke, a hematologist, came to the University of Kansas where he established the fact that platelets are reduced in purpura. He devised the famous Duke Bleeding Time test. He shortly became our first endocrinologist and then later an allergist. He had a favorite lecture on endocrinology. Once he was asked to speak to the Women's Christian Temperance Union in Kansas City. The meeting opened with a prayer and a violin solo, and the President of the WCTU then announced that Dr. Duke would speak on the subject of alcohol. Duke solemnly got up and said "I know nothing good about alcohol" and proceeded to give his favorite lecture on the glands of internal secretion. The lecture was accompanied by slides depicting men and women stark naked. The women demonstrated voluptuous breasts and the men had enormous and microscopic genitalia. The women in the audience were tremendously impressed, and it is said that this talk was the "talk of the town" for at least a month.

Another famous character in our medical school history was Dr. S. J. Crumbine, who was unfortunately rejected by the State of Kansas. He had practiced in Weir, Kansas, and came to the medical school to be Dean and Professor of Preventive Medicine. He was also the Secretary of the Kansas Board of Health which took most of his time and interest. He was a strong advocate of health through hygiene. It is said that he would board trains coming into Kansas and tear the common drinking cups off the wall. He popularized the slogan, "Don't spit on the sidewalk," which appears on souvenir bricks throughout Kansas. He initiated a campaign against flies. During his time a group of Boy Scouts fashioned fly swatters using a square of screen attached to a stick. He didn't know what to call this new gadget until he went to a baseball game where he heard the familiar "Swat that fly." This is said to be the origin of the fly swatter. Finally, there was mixed opposition to his crusades, and one day he found that his office door was locked and his key would not open it. So he said, "I guess it is time to leave Kansas." He went to New York City where he became well known nationally in the public health field.

Now let's change gears from ancient history to modern day history. There are a few professors among us who apparently have not reached sainthood yet, but who nevertheless have buildings named after them. Some of these were my first and favorite instructors. Dr. David Robinson was a resident in surgery when I was a student. I recall that following his first radical mastectomy he was very concerned that the results would be satisfactory. I, the student extern on the service, had to get up several times that first post-operative night to check on the dressings. From him I learned, "Show concern for your patients — even if someone else has to get up at night." From Mahlon Delp I learned of the physician's responsibilities to patients. There are so many wonderful stories about Mahlon Delp that it would be redundant for me to repeat them. Nevertheless, he is a scholar, and in a sense he was the lord of the wards. Another of my early instructors, Dr. Max S. Allen, will soon appropriately receive the University's highest honor — the Distinguished Service Citation.

Dr. Franklin D. Murphy was Dean of the medical school when I returned to join the staff in 1949. The administration building in the 1924 Bell Memorial Hospital is named for him. In our interview in 1949 in that building we talked briefly about the potassium ion and its importance to patients and research. I had just recently written a paper on the newly discovered importance of the potassium ion in refractory alkalosis in adults. In the same year another one of our Professors of Pediatrics, Dr. Daniel C. Darrow, wrote an important paper regarding potassium depletion in infants. Fortunately for me, Dr. Murphy hired me on the spot, for which I have been forever grateful. As all of you know, he went to the people of Kansas, primarily through the Kansas Farm Bureau, and put Kansas on the map with his preceptorship program and continuing education courses throughout the state. From him I learned, "Pay attention to Kansas farmers — they're the salt of the earth."

Dr. W. Clarke Wescoe came here as a young pharmacologist and soon became the dean of the medical school, and then the Chancellor of the University, just as had Murphy. He didn't take the Santa Fe Trail going west, but took his own trail back east and is now the chief executive officer of the Sterling Drug Company. Clarke Wescoe acted promptly and he told me that he never wasted time writing memos, and so I learned a basic lesson from him, "Never write a memo — it may be used against you."

Most of you know our good friend, Dr. Herbert

Miller. He came from Connecticut, and built a strong Pediatrics Department involving most of the pediatricians in metropolitan Kansas City. He was also instrumental in developing the new Mercy Hospital in Kansas City, Missouri. After many of his part-time pediatrics staff went to the new hospital in Missouri he retired and went back to Connecticut. An important building is named after him.

Now let's change gears again and return to my school days in the 1940s. The first professor I remember vividly was Dr. Joe Latimer, Professor of Gross Anatomy. You will remember that anatomy was taught both from the cadaver and from the book — and by osmosis. In the book, the arteries were colored red, the veins blue, and the lymphatics green. I can remember my frustration in dissecting the cadaver and not being able to find lymphatics — they were not colored green in the cadaver. I learned a lesson from that experience: "The addition of color to pictures may make it prettier — but not necessarily more accurate."

Another teacher during that first year of medical school was Dr. Henry Tracy, Professor of Anatomy, whom many of us will remember. The main recollection I have of his teaching consisted of a thoroughly preposterous statement he made one afternoon in class. He said that married couples usually have sex relations only about twice a week! I couldn't believe it, probably because I wasn't married at that time. After I was married I had a lot of fun developing a long term, randomized, uncontrolled, double blind study trying to disprove his statement. The results of that clinical investigation are here tonight in the persons of our children: Richard, Robert, Peggy, and Kathy. The co-investigator in this project was my wife, Beth. From Dr. Tracy I learned: "The word of the professor may or may not be the gospel truth."

One of my favorite professors was Dr. C. B. Francisco, Professor of Orthopedics during my senior year in Kansas City, the year after Beth and I were married. I don't recall much about his instructions concerning bones, but I do remember that it was at a class picnic in his yard that Beth told me she was pregnant. She told me she had been to see Dr. Harold Gainey that morning who confirmed the pregnancy. I was astounded, so I always associate that first pregnancy with Dr. Francisco even though he had nothing to do with it at all! Lesson no. seven therefore is: "If you're in trouble — don't blame it on the professor."

Dr. Logan Clendening is especially memorable to most of us. The beautiful fountain located in the courtyard in the heart of our Medical School complex typifies his vibrant spirit. Inscribed on this fountain is, "Life is short and the Art is long." Dr. Clendening came to the University of Kansas to teach Physical Diagnosis, became a book collector, historian, and a writer of therapeutic textbooks and a syndicated column. The Hixon Building on our campus was built with his wife's family's money. In this building the first Clendening History of Medicine Library was located. It was a beautiful and peaceful place in which to study. Dr. Logan Clendening and Dr. Ralph Major became fast friends and made the new History of Medicine Library one of the very best in the nation. Clendening's excellent teaching and his demonstrations of abnormal gait, for instance, stand out in our memory. He put his heart and soul into his teaching — and into his living. Once when the jack hammers got too loud in front of his home, he went into a rage, smashed up the machinery with an axe and got arrested and put in jail for it. From that experience I learned: "Don't put your heart, soul, or emotions into your teaching — you may end up in jail."

Dr. Arthur Hertzler and Dr. Ed Hashinger were contemporaries, but very dissimilar people. Dr. Hertzler was big and angular, the horse and buggy doctor. Dr. Hashinger was a sophisticated city collector of pressed glass. However, they have something in common. Dr. Hashinger would give almost every patient thyroid extract and Dr. Hertzler would take it away by total thyroidectomy and return the patient back to the endocrinologist for more thyroid extract. From that relationship I learned as a surgeon: "When in doubt, take it out — the internist may need another patient."

Many of us will recall Dr. Leroy Calkins. The new learning center is named after him. He was the second Professor of Obstetrics and Gynecology, after Dr. Don Carlos Guffy. The Home Delivery Program for medical students was in full swing at that time providing many memories for many of us. Dr. Calkins was a pragmatic teacher; he had very organized lectures. I had the impression that he could predict the moment of birth by numerical staging of labor which included his methods for counting the frequency and severity of contractions and putting numbers on the degree of cervical effacement. He was even skilled at estimating the amount of blood loss during labor down to the milliliter. I learned from Dr. Leroy Calkins a basic fact: "Put numbers on it — people may believe you."

Dr. Galen Tice almost singlehandedly ran the x-ray department, both the diagnostic and therapeu-

tic components of it. I was continuously amazed at his capacity for work.

Dr. Isenberger, Professor of Pharmacology, would often suggest that a certain drug, when injected into an animal, would cause the blood pressure to go up — or down — or both.

Dr. Earl Padgett was known for his capacity for work and spirits and for the Padgett-Hood Dermatome which was developed in Kansas. This device has revolutionized skin grafting in all parts of the world.

Don Carlos Peete is one of the kindest and most sincere professors at Kansas. He always knew the names of his students, even after many years of absence. From him I ought to have learned: "Try, at least, to know the names of your students!"

There are two skeletons which I have managed to dig up, as it were. One might be considered our temporary effort to teach medical students using the newly developed projected television techniques around 1950. Dr. Paul Schaffer, our Chairman of Surgery at the time, Dr. Orr, and myself were instrumental in this development. However we soon found that the "tail was wagging the dog" and that television was using the teachers instead of teachers using the television. Therefore, after about two years of teaching daily with television it became a skeleton in our curriculum. Another so called skeleton was the former use of rotation of students to the Osawatomie State Asylum and similar institutions. Many of us can recall those two week rotations there which in my instance, occurred shortly after Mrs. Friesen and I were married.

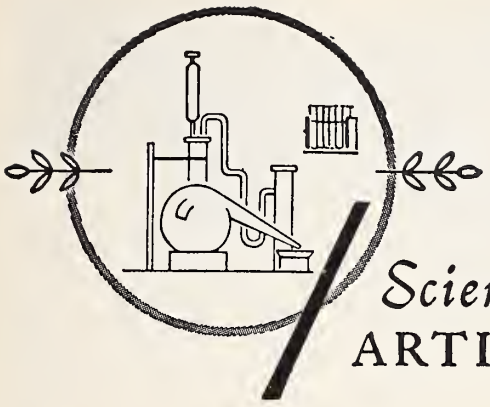
I wish now to pay tribute to the four professors at the University of Kansas School of Medicine who most influenced me. At the same time they took leading roles in the development of this school. All four came from Johns Hopkins University. Just as Johns Hopkins had its four horsemen including the venerable Welch, Osler, Halstead, and Sims; Kansas, too, had its four horsemen in Wahl, Major, Haden, and Orr.

Dr. Harry Roswell Wahl was a tireless worker for our school. When I was admitted to Medical School I received a letter which was the epitome of brevity — it said simply, "You are admitted to Medical School as of September." Dr. Wahl didn't have time for longer letters because he singlehandedly held down three top jobs in this school at the same time. He became active Dean after the dismissal of Dr. Sudler, but kept the position for 24 years during which time ten buildings were built. He was also Chairman of the Department of Pathology following Ralph Major and also assumed the responsibilities of Superintendent of the Hospital for which he volunteered at no pay. It now takes 62 professional people to do what he did singlehandedly in those days. From Dean Wahl I learned: "Don't depend on the bureaucracy — do it yourself."

Dr. Ralph Major, my idol, is both saint and scholar for most Kansans. He came from Johns Hopkins as the first pathologist in that dirty, dilapidated pathology building on Goat Hill. After a few years he went to Detroit, then returned to Kansas to be Chairman of the Department of Medicine. In 1922, Banting and Best discovered insulin; in 1923 Major's article on the clinical use of insulin in diabetics[1] was one of three important papers on that subject that year. It was a benchmark medical paper. This paper illustrates patients with diabetes before and after the use of insulin. Dr. Ralph Major was a superior physician. He was an example, following his own rule in which he said the physician should not only be a physician, but also an educated man. He was a scholar. His book, "Classic Descriptions of Disease," is a classic in itself, as are his books on the history of medicine and physical diagnosis. I learned many things from Dr. Ralph Major, one of which was an appreciation of the beauty of old books. Darwin has said, "If I had my life to live over again, I would make it a rule to read some poetry and listen to some music at least once every week — the loss of this happiness may be injurious to the intellect." In that respect, I learned from Major: "Try to read a bit each week — even if it is out of print." I learned also from taking care of him as a patient. I operated upon him four times. Each of three times was prompted by another emergency. I learned this lesson from him: "If you're unhappy — declare an emergency and act on it."

The postoperative experiences with him were fantastically interesting. In his delirium he would take me through his travels to Turkey, Egypt, and Italy. When his mind cleared postoperatively he was disappointed to find he was still in a hospital bed. From that I learned: "It's O.K. to dream a bit — but it won't get you anywhere." When he was our first pathologist here he told of taking two medical students out for a beer after performing an autopsy together in one of the funeral homes. The very next morning he was called on the carpet by Dean Sudler. The lesson that Major learned from that experience was: "News of beer drinking travels fast among students — avoid it." That attitude in 1915 is a far cry from today's when the medical students ap-

(Continued on page 165)



Duchenne's Muscular Dystrophy

The Diagnosis and Management of Cardiac Involvement

LEONE MATTIOLI, M.D.* ***and*** **MARK MELHORN, M.D.,** ***Kansas City, Kansas***

Two boys with Duchenne's muscular dystrophy (DMD) and congestive cardiomyopathy are presented and the literature is reviewed. Cardiac involvement occurs frequently in DMD and is best detected by an electrocardiogram. Early treatment with salt restriction and anticongestive measures may improve quality of life and reduce cost of care.

DUCHENNE'S muscular dystrophy (DMD) causes the myocardium to undergo degenerative changes similar to those affecting skeletal muscles. This process appears to affect nearly all DMD patients and may lead to disabling cardiac impairment. The diagnosis of cardiac involvement rests, classically, on electrocardiographic criteria.[1, 2] Approximately 97 per cent of children with DMD have abnormal electrocardiographic findings.[3] As many as 84 per cent of patients show myocardial involvement at autopsy.[4] Despite this very high incidence of cardiac involvement, symptoms of cardiac failure are rarely elicited and the most commonly cited cause of death in DMD is respiratory failure. This discrepancy may be due, in part, to the difficulty in diagnosing cardiomyopathy in patients markedly disabled by skeletal muscle abnormality, and usually confined to a wheelchair.

Described here are two boys with DMD and severe congestive cardiomyopathy, one of whom died. The cases clearly show the difficulties in recognizing symptoms of congestive heart failure in patients confined to a wheelchair and in managing this severe form of cardiomyopathy. In addition, the literature is reviewed to aid the physician to understand and deal with this ominous complication of DMD.

Case Reports

Case One: A 10-year-old white male with DMD since age 3 years and with persistent vomiting of one week's duration was referred to the University of Kansas Medical Center for a gastrointestinal work-up. He was confined to a wheelchair, and his daily activities consisted of wheelchair ambulation with assistance for transfer. In addition to vomiting, he had loss of appetite, a dry, weak and non-productive cough, headache, and his weight had increased 20 kg during the last 18 months. On admission (March 1980), blood pressure was 110/80; pulse rate, 92 beats/min; respirations, 28/min; temperature, 37.4C; and weight, 58 kg (greater than 100th percentile). Breath sounds were decreased bilaterally and symmetrically; no rales were heard. Pulses were somewhat reduced in volume, and there was a Grade II/VI apical systolic murmur. The second heart sound was normal. The abdomen was tense, soft, and obese. The liver and spleen could not be palpated.

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Figure 1a and 1b. Note progression of cardiac enlargement during a period of three years.

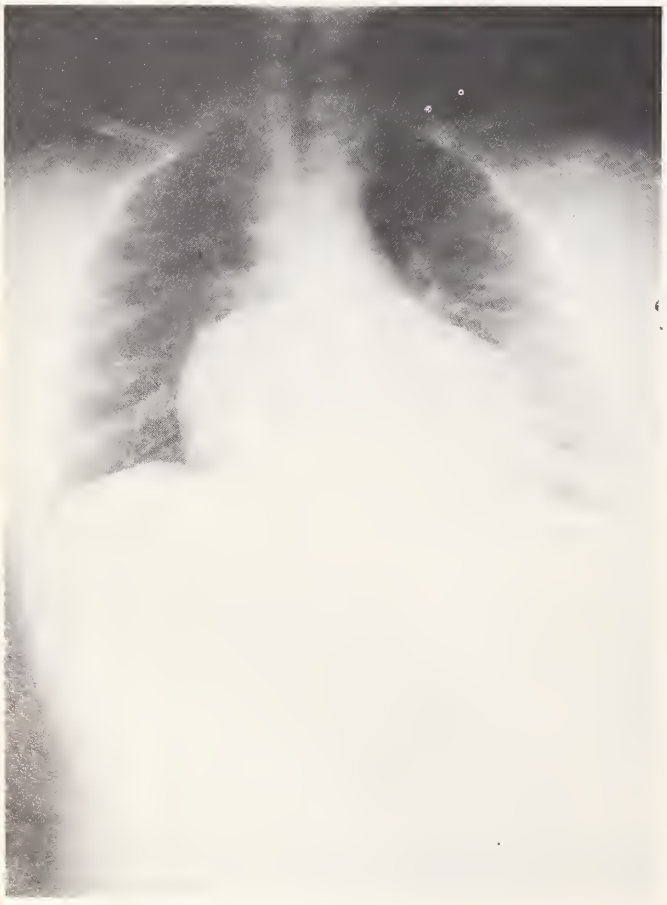


Figure 1. Chest x-rays in Case One.



Figure 1c. Note marked cardiomegaly with interstitial edema before treatment.

Figure 1d. Cardiomegaly remains unchanged after treatment.

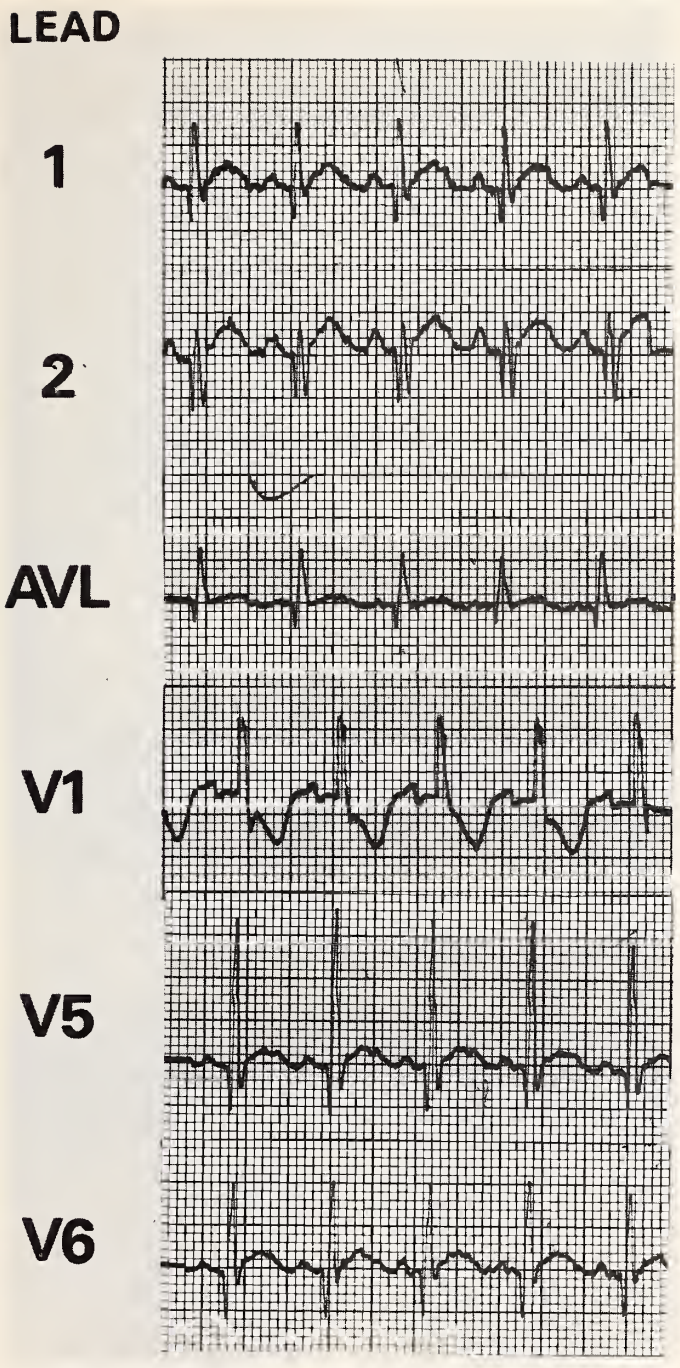


Figure 2a. EKG in Case One. Note deep Q waves in Leads I, II, V5 and V6, and the predominant R wave in V1.

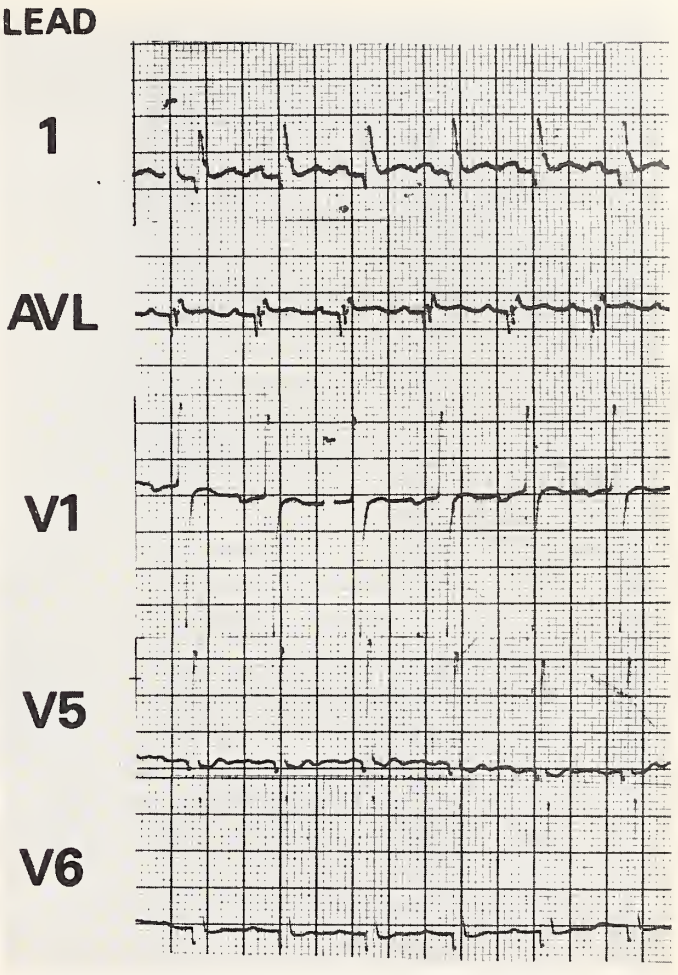


Figure 2b. EKG in Case Two. Note left atrial enlargement, slurred QRS complex in Leads I and AVL, deep Q waves in Leads I, AVL and V6, and flattened T waves in V5 and V6.

Chest x-ray *(Figure 1c)* showed cardiomegaly with interstitial pulmonary edema. Electrocardiogram *(Figure 2a)* showed left axis deviation, right atrial enlargement, and deep Q waves in Leads I, II, AVL, V5, and V6. The echocardiogram *(Figure 3a)* showed a markedly dilated left ventricle with a reduced posterior wall and septal motion. There was also a small amount of pericardial effusion. A review of prior chest x-rays showed the heart to be of normal size in November 1977 *(Figure 1a)* and moderately enlarged in March 1980 *(Figure 1b)* with some interstitial pulmonary edema.

Shortly after admission, the patient developed overt signs of pulmonary edema with wheezing, rales, gallop rhythm, and cyanosis. A prompt clinical response was obtained with intravenous furosemide and digoxin. A 7 kg weight loss occurred in two days. A repeat chest x-ray *(Figure 1d)* showed decreased pulmonary congestion without change in cardiac size. A repeat echocardiogram showed slight improvement in the left ventricular posterior wall and septal motion *(Figure 3b)*. The patient was discharged on the fourth day on digoxin, furosemide, and spironolactone.

He was subsequently readmitted to the hospital three times with bouts of pulmonary edema which

TABLE I
HEMODYNAMIC DATA IN CASE #1*

	Control	*Nitro-glycerine*	*Control*	*Nitro-prusside*	*Control*
Heart rate (beats/min)	130	140	130	130	120
Mean pulmonary arterial pressure (mmHg)	16	14	16	15	23
Mean aortic pressure (mmHg)	89	83	89	83	90
Pulmonary artery wedge pressure (mmHg)	7	5	7	7	11
Cardiac index (L/min/M^2)†	3.7	2.9(−22%)	3.4	2.8(−18%)	3.2
Stroke volume index (ml/beat/M^2)	28	21	26	22	27
Pulmonary vascular resistance (mmHg/L/min/M^2)	2.4	3.0	2.6	2.8	3.7
Systemic vascular resistance (mmHg/L/min/M^2)	23	26	25	28	27

* Data obtained after furosemide and digitalization.
† By thermodilution.

responded to intravenous furosemide. A right heart catheterization was performed to investigate the possibility of using peripheral vasodilators as adjuncts to the medical treatment of his congestive heart failure.[5] As shown in *Table I,* the pulmonary wedge pressure and cardiac index were within normal limits. However, the patient was able to maintain a normal cardiac index at rest by increased heart rate. Both nigroglycerin and nitroprusside reduced cardiac output; thus, neither drug could be used as an adjunct to treatment of congestive heart failure.

Twelve months following the initial episode of frank congestive heart failure, he is still alive, comfortable, with occasional exacerbation of pulmonary edema which responds well to intravenous furosemide.

Case Two: A 16-year-old white male with DMD was admitted to the University of Kansas Medical Center with a three-week history of dry, hacking cough and persistent vomiting. He had recently been treated with antibiotics for pneumonia, and was admitted on March 24, 1980, because of lack of improvement. On admission, he was coughing constantly. He was pale; blood pressure was 112/60; heart rate, 120 beats/min; respirations, 24/min; weight, 64 kg (70th percentile); and temperature, 36C. The first heart sound was muffled; the second heart sound was closely split. There was a Grade III/VI holosystolic murmur at the apex. There was an S_3 gallop, but no rales were heard. The abdomen was soft and nontender without organomegaly.

Chest x-ray showed cardiomegaly with interstitial pulmonary edema *(Figure 4c).* Electrocardiogram showed a normal sinus rhythm, left atrial hypertrophy, and deep Q waves in Leads I, AVL and V6, and flattened T waves in the lateral chest leads *(Figure 2b).* The echocardiogram showed a markedly dilated

Figure 3. Echocardiograms in Case One.

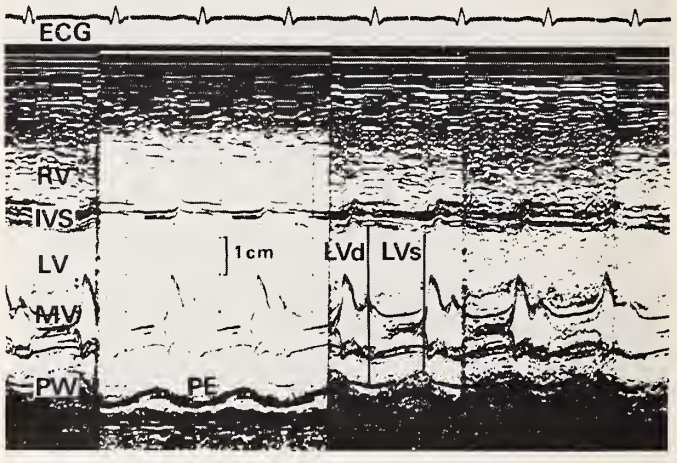


Figure 3a. Before digitalization. Note flattened septal (IVS) and posterior wall (PW) motion and reduced shortening fraction (LVd-LVs ÷ LVd). LVd = left ventricular diastolic dimension. LVs = left ventricular systolic dimension. PE = pericardial effusion.

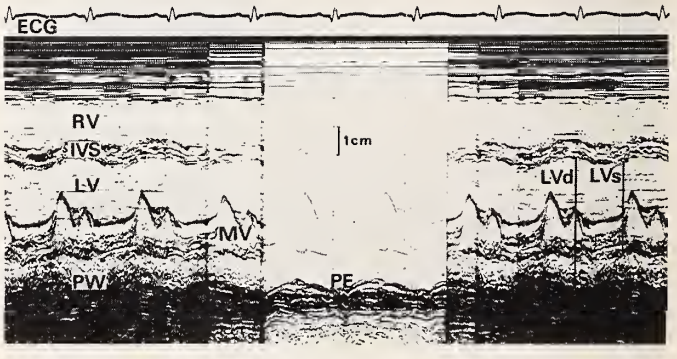


Figure 3b. After digitalization and diuresis. Note improvement in IVS and PW motion and in the shortening fraction. PE is also decreased.

Figure 4. Chest x-rays in Case Two.



Figure 4a. Normal heart size in January 1971.



Figure 4c. Cardiomegaly with interstitial pulmonary edema on March 24, 1980.



Figure 4b. Mild cardiomegaly with some evidence of interstitial pulmonary edema in January 1980.

left ventricle with reduced posterior wall and septal motion. A review of prior chest x-rays showed the heart size to be normal in January 1971 *(Figure 4a)*, and to be enlarged in January 1980, with some evidence of interstitial pulmonary edema *(Figure 4b)*.

Treatment for congestive heart failure was begun with furosemide and digoxin, intravenously. There was a short-lasting clinical improvement with the disappearance of the gallop rhythm; however, the heart rate remained persistently at 150 beats/min. The patient was discharged on digoxin, furosemide, and spironolactone after 12 days of hospitalization. At home, symptoms of pulmonary edema soon recurred. Prazosin was empirically added to the medical regimen. Two months after initial symptoms of congestive heart failure, he died.

Discussion

Pathology

Pathological features of cardiomyopathy in Duchenne's muscular dystrophy are atrophy of myocardial cells and replacement by connective tissue. A few mononuclear cells and fibroblasts are seen, a finding consistent with a non-inflammatory process. The proximal two-thirds of the posterior wall of the left ventricle are most commonly

affected. The left ventricular papillary muscles may be involved with resulting mitral valve prolapse and regurgitation. The mitral valve leaflet and chordae tendae are normal.

Electrocardiography

Several studies have confirmed that the electrocardiogram is a sensitive indicator in myocardial involvement in DMD. Distinctive electrocardiographic abnormalities include tall R waves or abnormal R-S ratio in V1, deep but narrow Q waves in the limb and left lateral leads, and ST-T segment variation. In addition, sinus tachycardia, multiple frequency notches on the QRS complex, and various forms of arrhythmias have been reported.[1-3, 5] It is agreed, however, that these patterns are not pathognomonic of the cardiomyopathy of DMD. Controversy exists concerning the development of these changes. Perloff *et al.*,[1] on the basis of autopsy findings in two cases, have suggested that the increased anterior forces reflect loss of potential generating myocardial cells in the posterior-basal portion of the left ventricle, and that the Q waves reflect extention of the process to the antero-lateral wall of the left ventricle. Ueda *et al.*[6] have added additional evidence that loss of posteriorly oriented forces causes anterior displacement of the QRS loop. However, this view is not supported in a much larger pathological study reported by Leth and Wulff.[3] A longitudinal study has been reported by Tanaka *et al.*[7] in 40 patients with DMD observed for an average of 5.4 years. In 75 per cent of these patients, based on the electrocardiographic criteria alone, the cardiac lesion appeared to have progressed from the basal portion of the left ventricle to the lateral wall of the left ventricle.

Most authors agree that there is no correlation between the electrocardiographic abnormalities and the degree of motor handicap and duration of illness. Electrocardiographic abnormalities similar to those observed in patients with DMD have also been observed in a small proportion of carriers.[8]

Echocardiography

Several echocardiographic studies have been reported in patients with DMD and in carriers. Sanyal *et al.*[9] have described mitral valve prolapse in 11 of 20 boys with DMD, all of whom had abnormal electrocardiographic findings. The authors postulated that mitral valve prolapse is the result of degenerative processes of the papillary muscle or the left ventricular myocardium. Biddison *et al.*[10] found a high incidence of mitral valve prolapse in carriers of DMD, and have suggested that echocardiography may be useful in identifying the coexistence of the dystrophic carrier trait if these findings could be confirmed.

Echocardiographic assessment of left ventricular function has also been reported in DMD. Ahmad *et al.*[11] have studied left ventricular function in ten children with DMD, ages 5-12 years, all of whom had electrocardiographic abnormalities. All systolic function indices were within normal limits. Diastolic function was also considered normal. The author concluded that overall ventricular function is well preserved in children with DMD despite EKG abnormalities. Farah *et al.*[12] have also studied 36 patients with DMD, 92 per cent of whom had electrocardiographic abnormalities. These authors found that the maximal diastolic endocardial velocity was reduced in 90 per cent of the patients, but there was a poor correlation between this abnormality and ejection fraction. The authors concluded that myocardial relaxation is abnormal in most patients with electrocardiographic abnormalities. However, there was no correlation between the severity of skeletal muscle disease and left ventricular dysfunction. In addition, although skeletal muscle disease progresses with age, there appears to be no concurrent significant progression in left ventricular dysfunction. Goldberg *et al.*[13] by echocardiography, have confirmed that left ventricular diastolic relaxation appears to be the major abnormality in DMD.

Tanaka *et al.*[7] have examined left ventricular function in three groups of children, age 6-16 years and older. Stroke volume index, ejection fraction, circumferential fiber-shortening velocity, systolic and diastolic posterior wall excursion, and left ventricular systolic time indices were compared in these three age groups. In Group 1 (age 1-10 years), only stroke volume index was significantly reduced. In Group 2 (age 11-15 years), all measurements except maximal rate of circumferential shortening were abnormal. In Group 3 (age 16 years and older), all indices of left ventricular function were abnormal. The authors concluded that left ventricular function may deteriorate during life with advancing age. More recently, Danilowicz *et al.*[14] have studied left ventricular function in a group of 36 patients, age 3-23 years, all of whom had electrocardiographic abnormalities. Only two of the 36 patients had normal left ventricular function. The authors concluded that echocardiography is an adequate tool in assessing cardiac function in DMD.

From these studies, it would appear that electrocardiographic changes are the earliest signs of myocardial involvement in DMD, and that echocardiography is an adequate technique to assess and

follow left ventricular dysfunction in DMD. Whether the cardiomyopathy in DMD is of a progressive nature remains to be clearly shown.

Clinical Features of Cardiomyopathy

Myocardial impairment in DMD remains clinically silent for many years, and the clinical diagnosis is delayed until overt signs of congestive heart failure appear. This is due primarily to absence of exertional dyspnea due to lack of activity.[15] Furthermore, radiographic evaluation of cardiomegaly is difficult due to poor inspiratory effort, obesity, elevated diaphragm, and thoracic deformity. When left ventricular function is severely compromised, as in our two patients, little improvement can be expected from anticongestive measures. Vasodilator therapy has recently been introduced in the treatment of congestive heart failure in addition to digitalis and diuretics.[16, 17] Although the conceptual basis for this therapy is sound, these drugs should not be used indiscriminately because they are experimental and there is little experience with their use in children.[18]

Based on the available information from the literature, it would appear that an electrocardiogram and an echocardiogram should be obtained when the diagnosis of DMD is made. Parents should be told of the cardiac involvement when EKG signs are present. Left ventricular function should be followed sequentially by echocardiography. Digitalis, salt restriction and diuretics may be instituted when echocardiographic signs of left ventricular dysfunction appear. This approach may delay symptoms of congestive heart failure and improve the quality of life for these unfortunate children. Whether this early supportive treatment alters the natural course of this cardiomyopathy remains to be demonstrated.

Summary

Two boys with DMD, congestive cardiomyopathy, and heart failure have been presented. Cardiac involvement in DMD occurs frequently, may be progressive, and has a poor prognosis. The clinical diagnosis of cardiomyopathy is made when late stages of cardiac decompensation occur because of lack of symptoms due to lack of activity. The electrocardiogram detects the earliest stages of the cardiac involvement, and the echocardiogram is a sensitive non-invasive tool to assess myocardial dysfunction. When signs of congestive heart failure are overt, the downhill course is rapid and little improvement can be expected from conventional decongestive measures.

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Artificial Respiration

A New Mode Utilizing High Frequency Positive Pressure Ventilation

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TERRESTRIAL animals were once divided by Krogh[1] into two groups: those that breathe by diffusion and those that employ mechanical respiration, the latter implying rhythmically recurring pressure gradients between the atmosphere and the intrapulmonary gases. Mechanical respiration provides a vast improvement in efficiency of gas exchange, and is essential to the maintenance of the high metabolic rates of mammals. As gases traverse the finer arborizations of the lung, however, respiratory movement is less distinct. The diffusion process becomes more predominant, and is fully operative at the alveolocapillary level. Great interest has recently been focused on the boundary conditions of pressure, volume, and flow that are essential to gas exchange in humans. In particular, the extent to which augmented diffusion respiration can be utilized has occupied the attention of both researchers and clinicians.

Draper and Whitehead (1944)[2] demonstrated that oxygenation could be satisfactorily maintained in the dog by diffusion alone. Carbon dioxide escape was too slow, however, and progressive respiratory acidosis developed. Obviously some ventilatory exchange had to be provided; the question was how much. Opinion at that time maintained that the tidal volume had to be larger than the deadspace volume; otherwise most of the ventilation would be deadspace ventilation and little gas exchange would occur. There were those who did not agree.

In 1955 the well known inventor and manufacturer of resuscitative devices, John (''Jack'') Emerson, applied for a patent for a high frequency ventilating device. The patent[3] describes this invention as ''an apparatus for treating a patient by vibrating a column of gas which is in communication with his airway, the column being vibrated at a rate which is greater than the patient's normal rate of inhalation and exhalation.'' Emerson's inspiration for employing this principle was his observation of the panting of a dog. He wondered how the animal could ventilate with volumes less than the anatomical deadspace. Apparently, he theorized, vibrating or oscillating the alveolar air could accomplish efficient gas exchange.

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High frequency positive pressure ventilation (HFPPV), a method of artificial ventilation in which small volumes of gas are introduced into the lungs at frequencies of 60-2400 cycles per minute, offers promise in the management of severe restrictive respiratory conditions and for ventilating patients with bronchial leaks. HFPPV can also be used to advantage in surgical procedures on the larynx and trachea.

Although Emerson aroused little or no interest in high frequency ventilation, the efficiency of small rapidly insufflated volumes of gas in washing out carbon dioxide was soon to be shown. Sanders (1967)[4] described the intermittent insufflation of jets of oxygen at fairly high pressures (20-50 PSIG) applied through the small delivery tube of a bronchoscope. This provided reasonably satisfactory gas exchange for brief clinical procedures. A similar technique, termed percutaneous transtracheal jet ventilation, was developed first for use in anesthesia[5] and later adapted for emergency resuscitation. A 14-16 gauge needle was introduced into the trachea via the cricothyroid membrane. Through this was passed a plastic cannula which was left indwelling, providing a conduit through which oxygen was delivered intermittently at pressures of 3-4 atmospheres. A more refined technique, suitable for use in microlaryngeal surgery, was described by Carden and Crutchfield (1972).[6] The cuffed terminal end of an endotracheal tube, bearing the oxygen jet tube secured by a silk suture, is introduced into the trachea below the larynx, and the cuff is then inflated. This provides a more stable positioning of the oxygen jet tube.

In 1970, a Swedish group reported experimental studies on positive pressure ventilation with small volumes delivered at frequencies of 60-100 cycles/

min.[7] They identified two clinical purposes that can be achieved with high frequency positive pressure ventilation (HFPPV). First, by means of endotracheal insufflation, a reduction in deadspace can be accomplished, and adequate ventilation can then be provided at lower airway pressures than those required by conventional intermittent positive pressure ventilation (IPPV). Second, with the short inflation times, the inertial characteristics of the lungs suppress the circulatory effects of the ventilatory pattern. In subsequent studies they demonstrated efficient maintenance of blood gas values at rates of 1000 breaths/min (BPM).[8]

HFPPV represents a radical departure from current concepts of mechanical ventilation. Conventional IPPV is delivered at normal respiratory rates (10-20 BPM), and alveolar exchange is intended to simulate spontaneous breathing. HFPPV on the other hand delivers small volumes of gas at rates from 60-2400 BPM (1-40 Hz).[9] At these rapid rates the concept that a given volume of gas is moved in or out during each breath no longer applies. It is impossible to distinguish inspiratory from expiratory flows at high frequencies, and they probably occur simultaneously.[10] The minute volume delivered by HFPPV must be larger than with IPPV, because a greater proportion is deadspace ventilation. Most importantly, intrathoracic pressures remain relatively constant at low levels, in contrast to the peak airway pressures of 25-35 cmH_2O observed with IPPV.

How carbon dioxide removal is accomplished is not fully known. The turbulence produced by intermittent jets of low volume (10-100 ml) is apparently sufficient to exchange gases.[11] It is also postulated that high frequency oscillations facilitate gas diffusion in the lung.[12] An explanation mainly based on the combined effects of diffusion and convection is offered by Slutsky *et al.*[13] Theoretical and experimental data advanced by these authors show that the significant variable determining the effectiveness of gas exchange is the amplitude of the oscillatory flow rate, independent of the values of frequency and stroke volume.

In HFPPV at rates of 100 BPM or greater, peak airway pressures range between 4 and 8 cmH_2O, and intrapleural pressure remains subatmospheric throughout the cycle. The adequacy of these low inspiratory pressures provides a method for ventilating stiff lungs,[14] and for management of patients with bronchial leaks.[15] It has been demonstrated that HFPPV can actually increase cardiac output when synchronized with the cardiac cycle. Also, HFPPV does not seem to cause the sodium and water retention reported with IPPV.[8]

HFPPV requires a mechanical ventilator with physical characteristics markedly different from machines that deliver IPPV. High inspiratory flows must be delivered at rapid rates. The response times and pressure/flow patterns generated by conventional IPPV machines are too slow to be practicable for HFPPV. This implies that not only must high flows be instantaneously initiated, but also that the internal volume of the ventilator circuit must be small, so that the compression volume is held to a minimum. Several models have been developed. One utilizes a piston driven pump; another incorporates a solenoid valve placed in line with a 0-60 PSIG pressure regulator.[10] Investigators, however, have focused their greatest interest on fluidic ventilators.[8] The Coanda or wall effect of fluidic logic systems makes it possible to construct a ventilator with minimum internal compliance, thus permitting rapid delivery of pressure and flow. A voluminous humidification system cannot be incorporated into the patient circuit; instead warmed humidified gases are delivered to the endotracheal tube via a catheter that is external to the system.

Alveolar collapse and progressive microatelectasis do not occur at HFPPV rates of 100 BPM or more. The functional residual capacity (FRC) is measurably increased and a small degree of positive end expiratory pressure (PEEP) is generated. Larger values of PEEP can be applied with HFPPV, and moderate increases do not appear to diminish venous return or cardiac output significantly.[8]

Weaning can be accomplished while HFPPV is continued. If the minute ventilation delivered by the HFPPV reduces $PaCO_2$ below threshold values, spontaneous respiration is abolished.[8] However, if the minute ventilation is gradually reduced, by decreasing the driving pressure of the inspired gas, spontaneous breathing is initiated when CO_2 accumulation begins to stimulate the brainstem respiratory centers. If HFPPV rates are well over 100 BPM, natural breathing can occur simultaneously with little or no patient discomfort.

The advantages of HFPPV may be summarized as follows. High intrathoracic pressures, as encountered with IPPV, are avoided, so that venous return is unimpeded and cardiac output is maintained. The FRC is sustained at normal values, or can be increased by PEEP, and distribution of gases is optimized. Weaning can thus be carried out without withdrawal of high frequency cycles. A number of clinical applications are suggested.

High frequency ventilation techniques for laryngeal and tracheal surgery are now well established.[8, 16, 17] Ventilation can be supported at low pressures, allowing the vocal cords to remain essentially motionless. HFPPV can be delivered through a very small bore endotracheal catheter, which can be introduced via the glottis, or transtracheally through the cricothyroid membrane. This type of insufflation technique has been advocated for emergency resuscitation. Cricothyroid insertion of a cannula is less difficult than endotracheal intubation under emergency conditions, and the high pressure jets of oxygen tend to prevent aspiration. HFPPV also seems particularly adaptable for treatment of restrictive lung conditions. Recent clinical reports indicate that HFPPV may be valuable for treatment of hyaline membrane disease of the newborn.[7, 18]

Some unanswered questions remain. One of considerable significance is determination of optimal HFPPV rate. Some clinical reports do not involve frequencies greater than 100 BPM. However, values of 500-2000 BPM have been satisfactorily utilized. There is some evidence that the efficiency of CO_2 elimination is decreased at rates of 900 BPM or above, a phenomenon termed "fade out" by Bird.[19] HFPPV will require much further study and critical evaluation, but already it has achieved limited acceptance as a treatment modality.

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Nominating Committee

(Continued from page 104)

1965. Practices Diagnostic Roentgenology. Has served as Councilor.

William K. Walker, M.D., Sedan. Born in 1928. Graduated from the University of Kansas School of Medicine in 1945. Is in Family Practice. Is now serving as Treasurer.

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Linda D. Warren, M.D., Hanover. Born in 1944. Graduated from the University of Kansas School of Medicine in 1970. Is in Family Practice. Is active in KMS and AMA Committee work.

Chronic Venous Ulcers

Techniques for Management

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Chronic venous insufficiency of the lower extremities is a common and distressing situation that is frustrating for both physician and patient. Edema, dermatitis, induration, pigmentation, and ulceration may be attributed to the post phlebitic syndrome,[1] although it is common to elicit no history of deep vein thrombophlebitis.

Venous hypertension in the superficial and communicating or perforating veins is the basis of the pathology. The usual venous drainage of the superficial tissue in the exercising leg in the erect position is toward the deep system via the perforating veins. The venous return in the legs is improved by the contraction of calf muscles, the so called "venous pump." Retrograde flow is prevented by competent venous valves in the deep and perforating veins. When these fail, the high pressure in the deep system is transmitted back to the perforating and superficial veins. This ambulatory venous hypertension is the basis of the syndrome of chronic venous insufficiency.

The most troublesome aspect of this condition is undoubtedly the venous ulcer near the ankle. The association of varicose veins and varicose ulcers was first described by Hippocrates, but there was not much progress made until Harvey discovered circulation. Gay[2] was first to describe the ankle perforating veins and introduced the word venous ulcer. In 1942, Bauer reported the high incidence of induration and later ulceration in patients following deep vein thrombosis.[3] After ten years, 72 per cent of patients had advanced stasis changes, and 52 per cent actually developed ulcers.

After venography was introduced, it was confirmed that following thrombosis the deep veins do recanalize, but the valves are indeed destroyed. As experience increased, Linton[4] emphasized the significance of the ankle perforators and the consequences of their incompetence. Crockett further described an ulcer precursor state, where a marked flare of dilated venules occurs around the medial malleolus due to the sustained local venous hypertension. The anatomy of the perforating veins in relation to the greater and lesser saphenous veins predicates the locations of the lesions in the so called "gaiter" area, the lower half of the leg extending upward from around the malleoli. The term, "Ankle Blowout" syndrome was thus introduced to the literature.[5]

Chronic venous insufficiency of the lower extremities has caused distress since ancient times. However, recently developed techniques for management, as described herein, can help to alleviate the manifestations of this condition.

The process starts when venous hypertension overcomes the osmotic pressure gradient and results in edema. The stasis phenomenon leads to decreased functional flow and impaired cellular metabolism. The supply of nutrients is diminished, with increased anaerobic metabolism and increased production of lactate, alanine, and carbon dioxide. This derangement in cellular activity results in cell death and tissue loss.[6]

Management

A good understanding of the abnormal physiology leads to a logical treatment plan. The main goal of treatment is the reduction of the local venous hypertension. In Owens'[7] experience, 85 per cent of cases of chronic venous insufficiency can be successfully managed on an outpatient basis. Therapeutic options are elevation, compression, and surgery. Elimination of infection and maximal healing of the ulcer are essential. Local antibiotics may be used after ascertaining the type of organism present in the wound. Some patients with chronic venous ulcers need small quantities of topical steroid creams to reduce the intense itching associated with the dermatitis. Whirlpool with betadine at neutral temperature has been helpful to reduce the bacterial flora of the base of the ulcer.

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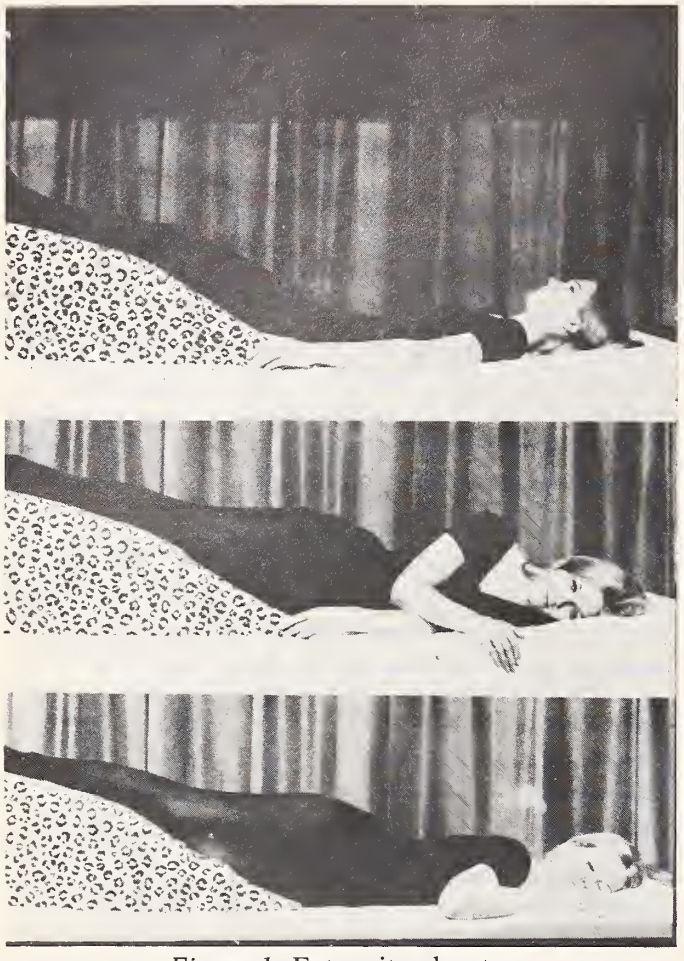


Figure 1. Extremity elevator.



Figure 2. Jobst inflatable sleeve.

Elevation

Although physicians advise their patients to elevate the involved lower extremities, compliance is often poor. In order to improve patient cooperation, Bernhard[8] designed a physiologically acceptable extremity elevator *(Figure 1)*. The following factors were taken into consideration: limbs must be elevated above the right atrium; there should be a 15 degree break at the knee to avoid the popliteal venous compression that can occur with full extension, and the elevator should be light weight, portable, and convenient for use in beds of any size.

Compression

Jobst intermittent compression machines can be used to reduce the venous edema of the lower extremities in a matter of days. Inflatable sleeves are put on the affected limb, and intermittent compression applied in a pressure gradient manner so that all fluid lodged in the tissues is milked out centrally *(Figure 2)*. The pressure applied and the duration of application can be varied, and depends on the patient's tolerance. This compression can be achieved on an outpatient basis and is very cost effective. The effectiveness of compression can be demonstrated by the measurement of the girth of the limb before and after compression. When no more reduction of the girth of the limb can be achieved, the limb should be measured for custom fitting of a Jobst garment. Jobst venous pressure gradient supports *(Figure 3)* are built so that the physician can select from a variety of counter pressure ranges and prescribe pressure gradient support with 25, 30, 40, or 50mmHg commencing at the ankle. They can also be made to fit one or both lower extremities. To produce a gradient support with predetermined circumferential pressure



Figure 3. Jobst venous pressure gradient supports.



Figure 4. Unna boot.

built in, this fabric has been developed with unidirectional tension elements accompanied by closely spaced, prestressed rubber cords. Patients should use these garments all the time, except when they are in bed or bathing.

Stasis Ulcer

The principles for managing ulcerations are basically the same as those for chronic venous insufficiency. In addition, however, epithelization must be hastened. This also calls for resolution of edema and control of bacterial flora. For ambulatory patients, pressure gradient dressing with Unnas paste bandage (Unna boot) has been used. The ulcer is cleansed in a whirlpool with betadine for 20 minutes followed by application of antibiotic sparingly. When applicable, an ointment containing steriod preparation may be applied to the surrounding skin surface area to control intense itching. The ulcer is covered by four to eight thicknesses of gauze, 1-2 cm larger than the ulcer. Unna paste gauze (Dome paste) is then applied *(Figure 4)*, just in front of the first metatarsal to just below the knee, cutting the roll frequently during application as dictated by the anatomy of the extremity. The boot is applied smoothly and in a pressure gradient manner. The pressure should be greatest at the ankle and least at the toes and below the knee. This dressing is changed in three to seven days, depending on ulcer size, depth, and the amount of discharge. After the ulcer heals, the patient should continue wearing pressure gradient garments. As the garments come in different counter pressures of 30, 40, 50, or 60mmHg, a decision must be made at the time of measurement. The counter pressure necessary to reconstitute the normal pressure gradient depends on many factors. Among these are the patient's occupation, the degree of limitation of motion of the extremity, obesity, and degree of patient cooperation that can be expected.

Followup visits are essential, and remeasurement should be done once every six months. Patients should wear their supports from the time of rising until they are ready to retire. They are asked to refrain from lifting more than 25-30 pounds, and stooping down on hands and knees. The patient is advised to report any recurrent trauma or ulceration immediately because 24-72 hours delay may mean weeks or months of morbidity.

Venous ulcers that resist all conservative methods may need ligation of perforators, vein stripping, excision of ulcer and skin grafting, or a combination of two or more surgical procedures.

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Ovarian Cancer

Recent Progress in Surgery and Chemotherapy

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OVARIAN cancer continues to be the most lethal common gynecologic tumor. Eleven thousand women die of this disease annually. Whereas routine screening for cervical and endometrial cancer have made these amenable to earlier diagnosis and treatment, 60-70 per cent of ovarian cancer continues to be stage III when initially detected. Even when the disease is detected in stage I, 20-30 per cent of patients die of their disease.[1] Recent advances in surgical staging and debulking procedures have improved the ability for combination chemotherapy to make an impact on response rates and very likely on survival rates. The surgical approach to ovarian cancer will be briefly discussed before the chemotherapy aspects, because the former may severely limit the latter.

Inadequate staging is one of the most frequent errors in the management of ovarian cancer. The surgeon need not be familiar with the details of staging in order to make an adequate incision and a careful evaluation and description of the findings. Assessment of the upper abdomen must be performed.[2] A frequent error is to confine the evaluation strictly to the pelvis. This is frequently done because of a small, low transverse incision. In a series of 100 patients with ovarian cancer, 83 per cent had incisions inadequate to evaluate the upper abdomen.[3] In the event the surgeon discovers ovarian cancer unexpectedly, a small Pfannensteil incision may be converted to a Maylard or Chernez for adequate evaluation and debulking. Extension of tumor into the upper abdomen makes it clear that local therapy to the pelvis is not adequate for cure.

Full evaluation for staging purposes includes pelvic washings, description of pelvis, diaphragm, liver, aortic lymph nodes, stomach, pancreas, colon, mesentery, and gutters. Biopsy or removal of the omentum should also be performed. Before a patient is classified as stage I or II, disease in the upper abdomen must be ruled out by random biopsies. If this assessment is not performed, the patient is assumed to be a stage III and is treated as such.

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An aggressive approach — including cytoreductive surgery and new drug regimens — have improved the survival rate as well as the complete remission rate. Response rates of 80-90 per cent are common, and complete remissions — as determined by second look operation — are increasing.

Debulking of the tumor has been shown to be one of the major advances in the treatment of ovarian cancer, and definitely improves the long range response to chemotherapy. If the tumor can be reduced to the largest implant being 2 cm, response and survival rates are much improved.[4] Sometimes this may not be technically feasible or the surgeon is caught unprepared with inadequate operative permits, blood, bowel preparation, or training. In that situation it is advisable to make a diagnosis and then arrange for more adequate surgical therapy at a later time. This is preferable to initiation of chemotherapy or radiation therapy after an inadequate debulking procedure.

Single agent chemotherapy has been the mainstay of therapy in ovarian cancer. Alkylating agents have proven to be more effective than the other groups of agents. Because these are easily administered, generally tolerated well, and give reliable and reproducible results of 40-50 per cent response rates, they are still frequently being used in ovarian cancer. Recent experience with new drugs and combinations suggest that there is now little place for single agent chemotherapy in ovarian cancer.[5, 6]

Combination chemotherapy attempts to take advantage of drugs of known effectiveness, different toxicity, and different mechanisms of action. Frequently they have more than an additive effect. Alkylating agents were first combined with antimetabolics with little increase in patient response. Adriamycin, which is effective as a single agent, was then combined with alkylating agents, with

TABLE I

PAC	Cisplatin, Adriamycin, Cyclophosphamide
CHAP	Cisplatin, Adriamycin, Hexamethylmelamine, Cyclophosphamide
CHAD	Cisplatin, Adriamycin, Hexamethylmelamine, Cyclophosphamide (Note dose is different than CHAP)
CHexUP	Cisplatin, 5-Fluorouracil, Hexamethylmelamine, Cyclophosphamide
Hexa CAF	Hexamethylmelamine, Cyclophosphamide, Methotrexate, 5-Fluorouracil
CP	Cisplatin, Cyclophosphamide

some improvement in response. Other agents were then combined, frequently producing more toxicity than increase in response.

Cis-dichlorodiammine platinum II (cis-platinum) was then used in patients with recurrent ovarian cancer. As a single agent it did not show improved effectiveness, but in combination with alkylating agents and other drugs it has shown a major effect on response rates and may well increase long range survivals. There are multiple combinations that appear effective as first-line combinations *(Table I)*. There is insufficient data to prove one regimen superior to the others. To be fully comparable, initial staging, grade, age of patient, residual tumor burden, performance status, toxicity, and method of assessment must be evaluated.

It appears from the literature that regimens including cis-platinum will produce an initial response rate of 70-90 per cent.[6-8] Progression-free survival is also improved. In our use of the combination of cis-platinum plus Cytoxan during the last two years we have had a 100 per cent response rate, when they were used as the initial chemotherapy. Whereas the one year survival rate with single alkylating agents is 50-60 per cent, we have had no deaths during the first year of therapy. Other reports from the literature have indicated that the rate of complete response is 40-50 per cent as determined by second look operations.[6-8] Our experience agrees with these figures. Survival data from second look procedures following single agent chemotherapy show that 70 per cent of patients with no evidence of disease at the time of surgery will be alive five years later.[4] Although there may be a gradual fall-out after this time, this would place the long range survival for stage III disease at 15-20 per cent. Time and numbers from our series and others are inadequate to confirm this. We currently have 24 patients under treatment, but longer followup will be required to determine long range surgival rates. We would expect certain subgroups, such as young patients with grade IMtumors and an adequate debulking procedure, to have a higher survival rate.

We also recommend combination therapy in stage II ovarian carcinoma in that 30-40 per cent of these patients die of their disease. Perhaps the only place for the single agent alkylating agent is in adequately staged, stage I tumors. It has been shown that stage 1A: G1 is the only category that does not profit from chemotherapy. As long as 20-30 per cent of stage I ovarian cancer patients do not live for five years, we must be aggressive with this group.

Tumors that continue to be confusing are those diagnosed as having low malignant potential. These may include stage III disease. Currently we treat these as invasive cancer if their biological course tends to so indicate.

The use of radiation treatment in ovarian cancer has declined during the last few years. Prerequisites for treatment include adequate staging, little residual tumor, and good facilities. Because of the nature of metastasis, total abdominal radiation to include the diaphragm must be used for best results. The dosage is limited by the tolerance of the small bowel, kidney, and liver. Radiation therapy has not proven to be more effective than alkylating agents and therefore combination chemotherapy appears to offer a better response. A true controlled study comparing radiation therapy against combination therapy has not been performed.

Part of the management of chemotherapy in ovarian cancer is the second look operation. Most centers have incorporated this into their followup. As the risks and side effects of the chemotherapy agents became more obvious, it became more important to terminate therapy if there was no evidence of cancer. The limitations of diagnostic procedures do not allow them to establish "no evidence of disease." For this reason a second exploratory laparotomy is advisable. Full evaluation of the abdomen is made to include biopsies and washings of pelvis, gutter, and diaphragm. The bowel is evaluated and aortic nodes sampled. If there is no evidence of disease, the chemotherapy may be terminated. This generally follows 12-18 courses of treatment.

(Continued on page 151)

Hyperprolactinemia

The Experience at the University of Kansas Medical Center

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The cases of 21 patients evaluated for hyperprolactinemia are discussed. The data reveal that patients with prolactin-producing pituitary adenomas have otherwise intact pituitary function. Sella tomography does little to distinguish patients with micro- and macroadenomas. In many cases, this form of amenorrhea can be successfully treated surgically.

HYPERPROLACTINEMIA has been increasingly recognized, in the past decade, as a common cause of amenorrhea and galactorrhea. This increased recognition is largely due to the availability of a radioimmunoassay for prolactin. In most cases, hyperprolactinemia is either drug-related, occurs postpartum (Chiari-Frommel syndrome), or is due to a pituitary adenoma (Forbes-Albright syndrome). A variety of commonly prescribed medications can induce hyperprolactinemia *(Table I)* and use of these drugs should be excluded prior to more extensive hormonal evaluation. The postpartum and tumorous manifestations are now felt to be subsets of the same disease; *i.e.*, both types of patient have either a micro- or macroadenoma.

Most pituitary tumors previously diagnosed as chromophobe adenomas have been reclassified as prolactinomas, and thus this type of tumor has become the most common pituitary neoplasm in adults. It has been estimated that about one-third of all patients with secondary amenorrhea and 75 per cent of patients with galactorrhea and amenorrhea have elevated serum prolactin levels.

During the past three years, we have evaluated 21 women with amenorrhea — with or without galactorrhea — and pituitary tumors at the University of Kansas Medical Center. Our experience is the basis for this report.

Clinical History

Table II summarizes the clinical features in 21 well studied cases. Galactorrhea was present in 18 (86%), which is similar to the data collected in one other series.[1] Two patients presented with primary amenorrhea whereas all others were secondary. Nine (43%) of our patients had a history of prior oral contraceptive use, and a total of 15 (71%) had taken estrogen either prior to the onset of amenorrhea or as a therapeutic modality to induce uterine bleeding. These findings are comparable to those previously reported.[2]

Four (19%) had significant hirsutism but none were virilized. None presented with visual field defects or signs of increased intracranial pressure. Two patients had significant headaches.

The mean duration of symptoms was 5.7 years with a range of 0.5-25.0 years. In all cases in which an adequate history could be reconstructed, the amenorrhea preceded the galactorrhea.

Endocrine Profile

A baseline pituitary hormone profile was determined for all patients. Testing included blood levels of trophic hormones and products of target glands

TABLE I
DRUGS KNOWN TO CAUSE HYPERPROLACTINEMIA

Estrogens
Phenothiazines
Tricyclic antidepressants
Butyrophenones
Minor tranquilizers
Antihypertensives
Cimetidine
Metoclopramide

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TABLE II
CLINICAL FEATURES

Duration of Symptoms (Yrs)	*Galactorrhea*	*BCPs Before Onset Sx*	*Hirsute*	*Sella Tomography*	*Preop* Prolactin*	*Postop Prolactin*	*Subsequent History*
11	+	−		N	64		Lost to followup
P	−	−	+	N	470		Lost to followup
8	+	−		N	247		Surgery not desired
2	+	−		N	100	8	Pregnant
2	+	−		N	730	132	Galactorrhea still present
6	+	−		A	99	5	Pregnant
10	+	+		A	98		Surgery not desired
15	+	−		A	315	80	Still amenorrheic
Unknown		−		A	89		Lost to followup
25	−	−		A	100		Surgery not desired
0.5	+	−		A	70	11	Menses resumed
1.3	+	−	+	A	115	40	Menses resumed
3.5	+	+	+	A	450	19.5	Menses resumed
2	+	+		A	165	100	Pregnant
2	+	+		A	500	4	Pregnant
3	+	+	+	A	660	120	Pregnant
2	+	+		A	190	20	Pregnant
2	+	+		A	82		Lost to followup
P	+	+		A	650	309	Good response/bromocriptine
2	+	+		A	80	9	Pregnant
4	+	+		A	71	16	Recent Surgery

A = abnormal. + = present. P = primary amenorrhea. N = normal. − = absent. * = normal prolactin <20 ng/ml.

(Table III). Prolactin levels were not significantly different in patients with and without galactorrhea. The mean value in the two groups was 260 ng/ml and 272 ng/ml, respectively (normal, < 20). The two patients with primary amenorrhea had a mean serum prolactin value of 639 ng/ml. All patients had normal serum cortisol and ACTH levels. Thyroxine values in all patients either were normal at presentation or were normalized by levothyroxine therapy, since it is known that primary hypothyroidism can indirectly induce hyperprolactinemia. Gonadotropin levels were all in the low to normal range, the latter being abnormal in view of the universally low serum estrogen levels in the amenorrheic patients. Three of 13 patients so studied had slightly elevated testosterone levels which correlated with the presence of hirsutism.

The patients were also subjected to various provocative and suppressive tests to assess the functional reserve of the pituitary gland *(Table III)*. All initially hypothyroid individuals were reevaluated after replacement therapy and these data are combined with the originally euthyroid group. Growth hormone stimulation tests were accomplished either with L-Dopa or insulin-induced hypoglycemia, and adequate stimulation was seen in 57 per cent. Thyrotrophin releasing hormone (TRH) infusion was used to stimulate thyroid stimulating hormone (TSH) and prolactin. Most patients not on exogenous thyroid had normal TSH responses (88%) but only 6 per cent of patients had a prolactin response to TRH. Prolactin suppression was tested using L-Dopa, and nearly all patients (95%) had normal decrements in prolactin levels. ACTH reserve was evaluated either with an overnight metapyrone test or insulin hypoglycemia, and all patients stimulated normally.

Radiologic Studies

Many of these patients were evaluated prior to the later generation of computerized tomography scanners. Sella tomography was done in all patients, and 16 had abnormalities in the sella contour while five

TABLE III
RESULTS OF SELLA TOMOGRAPHY

All Patients (N = 21)		*Abnormal Sella (N = 16)*	*Normal Sella (N = 5)*
Mean prolactin (ng/ml)	262	233	353
(Range)		(70-660)	(60-730)
Normal HGH stimulation	57%	50%	80%
Normal prolactin suppression	95%	93%	100%
TRH-prolactin stimulation	6%	8%	0
TRH-TSH stimulation	88%	92%	75%

were normal *(Table III)*. The two groups were quite similar, both in terms of prolactin levels and hormonal responsiveness.

Surgical Experience

Fourteen of these 21 patients underwent transsphenoidal hypophysectomy. Eight of the 14 (57%) had normal postoperative prolactins. The height of the initial prolactin elevation was of some importance in terms of the eventual outcome. The mean prolactin in the group that normalized was 211 ng/ml whereas patients who were still hyperprolactinemic had a mean prolactin of 439 ng/ml. However, the preoperative level was an inconsistent indicator of surgical outcome in the individual patient as the range of values was 70-500 ng/ml in the normalized group and 115-730 ng/ml in the hyperprolactinemia group. One of two operated patients with a normal sella, and six of 11 patients with abnormal sellas have normal prolactin levels. To date, in all patients in whom the prolactin value returned to normal it has remained that way.

Five patients with "normalized" prolactins have become pregnant by design. The other three have resumed menses but are not yet pregnant. Two additional patients with elevated prolactin levels (100 ng/ml and 120 ng/ml, respectively) have conceived with subsequent bromocriptine therapy. On this drug their prolactin levels decreased to 9 ng/ml and 15 ng/ml, respectively.

There were no serious sequelae to surgery. Several women had mild transient diabetes insipidus but in no case was this persistent. There were no instances of cerebrospinal fluid rhinorrhea.

To our knowledge, seven patients have not had surgery. Four were lost to followup; three patients did not desire surgery and are being followed.

Discussion

Hyperprolactinemia is becoming an increasingly frequent endocrinopathy. Most patients with persistent elevations of serum prolactin will present with amenorrhea and galactorrhea. Headaches, although infrequent in our series, have been reported in up to 25 per cent of patients.[1] Hirsutism is not a common accompaniment of the hyperprolactinemic state, and our incidence of 19 per cent far exceeds other published studies. While there is some evidence of direct prolactin activity at the adrenal level stimulating production of weak androgens such as dehydroepiandrosterone, the topic is controversial.[3]

Only about one-half of the patients had a growth hormone response to L-Dopa but this finding must be tempered by the lack of correlation between responsiveness and eventual outcome. Moreover, human growth hormone (HGH) responsiveness may be impaired under a variety of circumstances, probably the most common of which is obesity. Some patients do not respond well to certain HGH-provocative tests while they do to others, and this occurs in an entirely unpredictable fashion. Thus, a negative test is not very useful whereas a good response preoperatively is assurance of intact trophic hormone function.

Prolactin suppressibility with L-Dopa appears to have no diagnostic significance. More than 90 per cent of patients had no prolactin response to TRH infusion. However, our findings indicate that a normal test does not rule out the possibility of prolactinoma minimizing the role of this particular study in future patients. TSH responsiveness to TRH infusion was tested to exclude subtle primary or secondary hypothyroidism. ACTH stimulation tests need to be performed to exclude secondary adrenal insufficiency. Our suggested endocrine evaluation protocol is contained in *Table IV*.

Radiologic assessment of this condition is equally important. The value of sella tomography in assessing presence or absence of tumor is questionable. Five of our tumor patients had normal sella tomography. On the other hand, irregularities in sella contour are common in the healthy population. Twenty-three per cent of the normal population have tomographic evidence of a microadenoma, but in more than three-fourths of these individuals, no histopathologic pituitary lesion can be found.[4] Computer-

TABLE IV
SUGGESTED ENDOCRINE PROFILE

Hormone Determinations — Baseline
- Prolactin
- FSH
- LH
- Thyroxine
- Cortisols, both morning and evening
- Testosterone

Hormone Determinations — Stimulation
- TRH stimulation of TSH
- L-Dopa stimulation of HGH (or insulin hypoglycemia)
- Metapyrone stimulation of ACTH

Special Situations
1. IF hirsute, obtain serum androstanedione, DHEA-S
2. IF diabetes insipidus suspected, do water deprivation study

ized tomography with coronal cuts utilizing intravenous contrast has facilitated delineation of small tumors that have not caused sella erosion. This technique also obviates pneumoencephalography as it can identify both suprasellar extension and empty sellas. The diagnosis of an empty sella can best be confirmed using metrizamide cisternography in conjunction with CT scanning. We recommend CT scanning with axial and coronal sections utilizing intravenous contrast agents in all patients.

Although some surgical series indicate cure rates in excess of 80 per cent with the transsphenoidal approach,[5] most predict resumption of menses and normal prolactin levels of 40-60 per cent. The correlation between the height of the serum prolactin and the surgical outcome reflects the conclusion that larger, bulkier tumors are generally less successfully removed; however, even greatly elevated prolactin levels in no way preclude a good surgical result.

Some patients question the need for surgery. If galactorrhea is not significant, pregnancy not desired, and the uncertainty of amenorrhea is not disconcerting, there might be a tendency to observe these patients. There are certain hazards of nonintervention, and these include: (1) possible heightened aggressiveness of the tumor with resultant visual field impairment and pituitary insufficiency; and (2) estrogen deficiency leading to decreased bone mass.[6] We observed this latter complication in one of our patients with primary amenorrhea who had rather severe premature osteoporosis.

It can also be appreciated that the resultant infertility from these tumors is often reversible. Successfully operated patients must be motivated not to use estrogens subsequently for contraception due to the possibility that these agents might promote growth of the residual prolactin-producing cells.[7]

None of our patients in whom the prolactin level returned to normal have had recurrent hyperprolactinemia. These patients have now been followed for periods ranging from two months to three years. Nine patients in one recent report, who had normal prolactins following surgery, later developed recurrent disease.[8] In this latter study, the eventual outcome of surgery was not apparent until six months postoperatively at which time serum prolactin levels seemed to stabilize. Patients in whom the prolactin level remains persistently elevated can be treated either with dopaminergic drugs such as bromocriptine or with pituitary irradiation.

Bromocriptine (Parlodel) can be safely used in the operated patient with persistent hyperprolactinemia who desires pregnancy. Use of the drug in place of surgery cannot be endorsed at the present time due to insufficient data concerning the effects of pregnancy on the tumorous gland. For example, in one study from the Netherlands, three of nine hyperprolactinemic women who became pregnant with bromocriptine therapy alone had marked tumor expansion between the 22nd and 24th weeks of pregnancy.[9] Such rapid tumor growth may necessitate emergency surgery to relieve optic nerve compression, hydrocephalus, or more comprehensive pituitary failure. While the question of teratogenicity of bromocriptine is still not settled, the largest series reported failed to find any general increase in the fetal malformation rate[10] or evidence of a specific malformation profile, as for example was seen with thalidomide. If pregnancy is not wanted, pituitary irradiation would be the preferred treatment modality, since there is no firm evidence that bromocriptine has an intrinsic tumoricidal activity; hence discontinuation of the drug usually results in recurrent hyperprolactinemia. Then, too, the drug is not without side effects and it is expensive. One additional consideration is that bromocriptine should not be used without an extensive endocrine evaluation due to the multiple other causes of hyperprolactinemia, such as primary hypothyroidism and acromegaly. For these reasons, we feel the initial management of patients with prolactinomas should be surgical, with radiation and drugs used for surgical failures.

Conclusion

Patients with hyperprolactinemia need thorough evaluation of their hormonal status. When a tumor is detected, surgery can be performed with little risk to the patient, and with a good chance of restoration of menses, cessation of galactorrhea and, ultimately, fertility. When the prolactin level does not fall to normal, bromocriptine may successfully lower the level so that ovulation may occur with little concern about tumor expansion during pregnancy. Late recurrences should probably be treated with irradiation.

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Fluoridation and Cancer

An Epidemiologic Appraisal

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ARTIFICIAL fluoridation of public drinking water began in Grand Rapids, Michigan in 1945. Today, nearly one-half of the population of the United States has access to significant levels of fluoridation with 94 million people receiving artificial fluoridation. In Kansas, approximately 50 per cent of the state's population receives artificially fluoridated water.[1] However, Wichita, the largest city, is a major exception.

Despite its noted low cost and high effectiveness, the extent of fluoridation has been held up in part because of allegations that fluoride causes cancer. The National Health Federation (NHF) is partly responsible for this claim. In early 1979, representatives of this group appeared before the City Commission in Lawrence to urge the elimination of fluoride from the drinking water. Lawrence water has been fluoridated since 1953, and to date the citizenry has been exposed to more than 1 million person-years of exposure to fluoride at 1ppm. Yet Douglas County's cancer mortality rates are relatively low in comparison both to US and Kansas rates. In the end, the commissioners ruled that a non-fluoridated water tap would be installed at the water treatment plant so that those wishing non-fluoridated water could get it. This was a victory of sorts for those advocating freedom of choice. As far as the scientific evidence was concerned, the commission did not concur with the testimony of the NHF and others that fluoride causes cancer.

In October 1962, the water supply in Kansas City, Missouri, was fluoridated. However, in August 1964, a referendum was held and the anti-fluoridationists won by close to 5,000 votes (74,000 votes cast). The negative vote may have been partially due to fear that the addition of fluoride to drinking water might cause illness. The Supreme Court of the State of Missouri ruled that this public health issue could be decided by the electorate. Hence, fluoridation was stopped. In 1980, the City Council of Kansas City, Missouri again voted to fluoridate. The NHF and others objected and obtained sufficient signatures to again place the issue on the ballot, and this time the voters chose to fluoridate. However, after the election, the NHF requested a temporary injunction and a court hearing was held in April 1981. The judge held that the matter had been duly settled in an appropriate manner, and dismissed the case. Fluoridation was then reinstituted.

Evidence presented to date indicating that artificially fluoridated drinking water causes cancer has been examined, and a critique of the methodology and conclusions of those claiming a positive association between fluoride and cancer is offered. It is concluded that current evidence does not support the hypothesis that fluoride causes cancer.

The controversy caused by this 35-year-old public health practice continues. The purpose of this paper is to examine some of the cancer evidence previously presented by the NHF, list some of the negative results reported by other investigators, and to present some cancer data for Kansas. This information is presented from the viewpoint of an environmental epidemiologist.

The Anti-Fluoridationist's Argument

The principal argument that fluoride causes cancer is based on an analysis by Yiamouyianis and Burk.[2] The ten largest US cities that began fluoridation prior to 1960 (actually 1952-1956) were matched to the ten largest cities that were: (1) non-fluoridated as of 1969, (2) had comparable cancer death rates (CDRs) to the fluoridated cities 1940-1950, and (3) had CDRs exceeding 155/100,000/yr in 1953. Approximately 18 million people lived in the two groups of cities *(Table I)*. Inasmuch as New York City fluoridated in 1965 and Detroit fluoridated in 1967, they were excluded from the non-fluoridated group.

The key argument of the anti-fluoridationists is based on a graph showing the change in the mean CDRs from 1940 to 1970 for both fluoridated and

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TABLE I
FLUORIDATED AND NON-FLUORIDATED CITIES IN NATIONAL HEALTH FEDERATION STUDY

Fluoridated	*Non-Artificially Fluoridated*
San Francisco	Los Angeles
Washington, DC	Atlanta
Chicago	New Orleans
Baltimore	Boston
St. Louis	Kansas City, MO*
Buffalo	Newark
Cleveland	Cincinnati
Philadelphia	Columbus
Pittsburgh	Portland
Milwaukee	Seattle

* Fluoridated between 1962 and 1964.

non-fluoridated cities. From 1940 to 1950 the CDRs increased together at approximately the same rate. During this period the rates are fairly close together. About 1952, the rates start to separate, with the rates for fluoridated cities continuing on at about the same slope while the rates for non-fluoridated cities seem to level off for about ten years, then increase again at about the same slope. The result is that the rates for fluoridated cities are higher than the rates for non-fluoridated cities in 1970. While the original analyses did not consider age groups separately, newer analyses consider ages 0-24, 25-44, 45-64, and 65 and over. The increased CDR is most pronounced in the older two age groups.

The NHF argues that this graph, plus their ancillary data, prove that fluoride causes cancer. According to their argument, the only major event occurring in the fluoridated cities during the period 1952-1956 was water fluoridation. How well does this argument hold up against generally accepted principles of epidemiology? This question can be broken down further. Does their data show an association between fluoridation and cancer? If an association is found, can a causal link be demonstrated?

Association of Fluoride With Cancer

The cities selected for the two comparison groups are not matched or evaluated with respect to such variables as diet, education, socioeconomic status, ethnicity, age, sex and race structure, occupation, and air and water pollution. These factors can change cancer outcomes and are also changing with the passage of time. Many of the fluoridated cities were in older parts of the US (*i.e.*, the northeast and middle west) while many of the non-fluoridated cities were located in the southwest and qualify as sunbelt cities. It is very difficult to compare such cities as St. Louis, Baltimore, Buffalo, and Cleveland with Los Angeles, Houston, San Antonio, San Diego, and Atlanta. The southwestern cities were rapidly growing during the 1950s due to immigration from the frostbelt, and this could partially explain the flattening off in the rate curve for non-fluoridated CDRs. Indeed, this curve seems to show a continuing growth in CDR for fluoridated cities at almost the same slope as previously. It is the non-fluoridated cities that show a sharper decline in slope. The impact of leaving out New York and Detroit from the non-fluoridated group is uncertain.

For proper analysis, data need to be specific as to sex, race, and cancer site. Age specific or age adjusted rates are acceptable. The NHF's studies are not simultaneously sex and race specific and age adjusted. Using total cancer as the only index is invalid. For cancer in males, the major sites of concern are lung, prostate, and colon. For cancer in females, the major sites of concern are breast, colon, and lung. Each of these sites has separate risk factors. As far as external agents are concerned, lung cancer is predominantly related to cigarette smoking. Inasmuch as the anti-fluoridationists have admitted that lung cancer is unlikely to be caused by fluoridation, they should have removed this outcome from the analysis. Presumably, kidney, stomach, bone, and bladder are the major cancer sites of concern based on route of exposure and the toxicology of fluoride.

Another problem with the anti-fluoridationist's arguments relates to the latency period for an effect. The NHF states that the cancer effect occurs within one or two years. In contrast, the latency period for asbestos related lung cancer is 15-20 years. The latency for A bomb related leukemia in Japan was four to five years at a minimum. Thus, fluoride would cause a more sudden onset than either the A bomb or asbestos exposure in mining, milling, or insulation workers.

It would also be desirable to establish some estimate of the impact of dose-response as measured by person-years exposure. The NHF data do not provide an analysis for such an effect. For example, if city A has exposed 100,000 people for 10 years, that would be equivalent to 1,000,000 person-years exposure. On the other hand, if city B has exposed 100,000 people for 20 years that is equivalent to 2,000,000 person-years at risk. If fluoride did cause cancer, one would expect the cancer rates to be higher within fluoridated cities with a longer exposure period and with more person-years at risk (*i.e.*,

greater in city B than in city A). In addition, one would expect that rates would be higher in fluoridated cities as compared to non-fluoridated cities.

Causality

Thus, the NHF study failed to demonstrate an association, much less causality. Association needs to be established first. However, even if an association were present, several additional steps are necessary before it can be concluded that causality exists. To begin with, death certificates would need to be validated. The underlying cause of death as listed is notoriously imprecise. Incidence data are preferable to mortality data because they are a better measure of risk, particularly for cancer with a low case fatality rate. Thus, it would be useful to use data from existing state or city SEER registries (Surveillance, Epidemiology and End Results Program of the National Cancer Institute).

Finally, ecological studies are useful in generating hypotheses but even if they find positive associations, additional studies (such as case/control) need to be done to rule out such factors as diet, alcohol, smoking, and occupation. It is part of the responsibility of environmental epidemiologists to pursue such leads.

One criterion often applied to determine causality is whether an association observed is consistent with results found elsewhere. Seldom is the result from one study so overpowering that independent corroboration is not asked for. Research studies done by other independent investigators need to be considered. In the case of the NHF studies on fluoride and cancer, the unanimous opinion of the scientific community is that the methods used were invalid.

Substantiation of Research

A study by the National Cancer Institute (NCI) reviewed cancer mortality rates for 35 cancer sites in naturally fluoridated counties in Texas (Hoover *et al.*[3]). Counties were grouped from low to very high fluoridation levels. The study was controlled for age, sex, race, urbanization, and socioeconomic status. NCI found no evidence of excess cancer mortality as fluoride levels increased. There was no evidence of a dose/response relationship. NCI also looked at cancer mortality in all US artificially fluoridated counties, where more than two-thirds of the population was exposed. No excess was found as compared to non-fluoridated cities. Finally, NCI compared the incidence of cancer in fluoridated Denver, Colorado with the incidence in non-fluoridated Birmingham, Alabama. Denver was 66 per cent fluoridated by 1955. The results of this comparison showed no excess cancer incidence in fluoridated Denver. Thus, there is no consistent evidence of any differences between areas that could be related to fluoride. Incidence data give the same answer as the mortality analysis.

In another study by the NCI, the original data used by the NHF was reanalyzed to include population density, socioeconomic status, ethnic composition, percentage of population employed in manufacturing industries, and geographic location. The result was that only stomach cancer for males remained elevated. This one positive finding could be due to statistical association.

Rogot *et al.*[4] studied mortality trends from all causes in 473 US cities for the period 1950-1970. He specifically evaluated heart, kidney, and cancer mortality with respect to fluoridation status. The fluoridated cities showed an 8 per cent increase in cancer mortality ratio while the nonfluoridated cities showed a 7 per cent increase. His overall conclusion was that there are no consistent relationships between fluoridation and changes in either cancer, heart, or general mortality rates during the 20 year period.

Erickson[5] evaluated 24 fluoridated and 22 non-fluoridated cities from 1969-1971. He analyzed for all causes of cancer in blacks and whites. When data were adjusted for age, sex, race, population density and median education, there was no evidence of a harmful effect, including cancer, that could be attributed to fluoridation.

Oldham and Newell[6] reported that the two groups of cities in the NHF study differed in their age-sex-race structure in 1950. In addition, fluoridated cities had an excess of cancer deaths of 10.3/100,000 population in 1950. In 1970, the demographic structure differed much more than it had in 1950. The fluoridated cities had many more non-whites. When demographic changes were considered, they found that the excess cancer rate increased by 1 per cent during 20 years in the fluoridated cities and increased by 4 per cent during 20 years in the unfluoridated control cities. They conclude that there is no evidence relating fluoridation of US water supplies with cancer.

Taves[7, 8] evaluated cancer mortality from ten sites (mouth and throat, esophagus, stomach, colon, rectum, breast, ovary, kidney, bladder, and bone). He calculated standardized mortality ratios (SMR) for the periods 1949-1951, 1959-1961, and 1969-1971 in the next ten largest fluoridated cities (left out of the NHF study) and the five non-fluoridated cities omitted from the 15 largest non-fluoridated cities. He found no difference in SMR between the fluori-

dated and non-fluoridated cities.

Doll and Kinlen[9] evaluated the NHF analysis and said that when age, sex, and ethnic status are considered, the ratio between observed and expected cancer mortality declined in the fluoridated cities and did not change in the non-fluoridated cities. The standardized mortality ratio was greater than 1.0 in both sets of cities for 1950, 1960, and 1970. Doll makes the point that crude rates are useless. In addition, he has found that the proportion of the non-white population and the proportion of the white population age 65 years and over increased more rapidly in the fluoridated than the non-fluoridated cities.

Thus, the uniform conclusion of other scientists is that the NHF's study methodology is invalid; its study was not conducted in a method considered to be generally acceptable epidemiologic research.

Kansas Data

Recently, cancer mortality rates in white male and female residents of Kansas for the period 1973-1977 have been determined. Such rates are compared herein with fluoridation status for the counties with the largest population. The protocol used is as follows:

1. The county must have a white population ≥100,000 in 1970.
2. ≥75 per cent of the county's population must live in cities served by artificially fluoridated water (at 1ppm).
3. The county must have been fluoridated prior to 1970.
4. As an alternative to #2 and #3, a control county will be selected where ≥35 per cent of the county's population lives in cities that do not artificially fluoridate their drinking water.

Fluoridated counties selected include Shawnee, Johnson, and Wyandotte. The unfluoridated county is Sedgwick, which includes the state's largest city, Wichita. The average level of natural fluoride in Wichita's water is about 0.32ppm (Kansas State Department of Health and Environment).

Table II indicates the counties involved, the 1970 total white population, the percentage of change in white population from 1970 to 1975, and the minimum percentage of the white population receiving fluoridated water. The total white population exposed to artificial fluoridation is 512,393. Although Johnson County is closest to Sedgwick in terms of population, Shawnee County is closest in terms of per cent of change in the white population from 1970-1975. Wyandotte County may be closest in terms of the degree of industrialization.

Table III indicates the counties involved, the person-years (and total years) of exposure to artificial fluoridation until 1977, and the five year age adjusted cancer mortality rates (±1 standard deviation) for whites. Nine cancer sites are indicated that might possibly be related to either a waterborne carcinogen or specifically to fluoride. Due to its rarity, bone cancer was calculated by county group, with Johnson and Wyandotte counties combined.

As far as statistically significant excesses in fluoridated areas are concerned, Shawnee County is significantly elevated above the state rate for bladder cancer in males. Johnson County has no significantly elevated cancers. Wyandotte County is significantly elevated above the state rate for total and colon cancer in males and total cancer in females. Sedgwick County is significantly elevated above the state rate for total cancer in males. In comparison to

TABLE II
KANSAS COUNTIES SELECTED FOR COMPARATIVE ANALYSIS

	County	*1970 White Population*	*% Change in White Pop. 1970-1975*	*Minimum % Pop. Receiving Fluoridated Water*
Artificially fluoridated	Johnson	219,402	+7.8	89.2
	Shawnee	142,920	−3.2	79.1
	Wyandotte	150,071	−6.6	90.7
Fluoridated total		512,393		
Non-artificially fluoridated	Sedgwick	319,222	−2.1	—
Grand total		831,615		

TABLE III

1973-1977 Five-Year Cancer Mortality Rates (±1 S.D.), KANSAS WHITES (AGE ADJUSTED/100,000)

County	Person-Years (Total Years) of Exposure	Total M	Total F	Stomach M	Stomach F	Colon M	Colon F	Rectum M	Rectum F
Shawnee	2,261,240 (20)	847.2 ±31.4	604.46 ±25.73	23.7 ±5.65	22.07 ±4.94	83.61 ±10.6	82.73 ±9.54	8.40 ±3.33	11.83 ±3.13
Johnson	2,255,784 (11-17)	818.9† ±26.4	588.98 ±21.76	33.52 ±5.67	7.48 ±2.56	62.55 ±8.04	65.77 ±7.45	15.87 ±4.30	10.56 ±3.17
Wyandotte	1,783,759 (13-17)	1,063.8*‡ ±33.8	661.72‡ ±26.21	37.66 ±7.34	11.54 ±3.48	110.98‡ ±12.17	82.14 ±9.34	27.18 ±6.23	10.10 ±3.22
Sedgwick	-0-	916.4‡ ±22.3	593.87 ±17.27	27.31 ±4.4	13.56 ±2.59	77.49 ±7.28	69.91 ±5.91	20.21 ±3.75	9.99 ±2.20
STATE	N/A	840.50 ±7.50	583.45 ±6.28	32.19 ±1.60	14.13 ±1.00	77.33 ±2.42	74.73 ±2.21	19.33 ±1.23	12.53 ±0.89

County	Person-Years (Total Years) of Exposure	Liver M	Liver F	Pancreas M	Pancreas F	Kidney M	Kidney F	Bladder M	Bladder F	Bone§ M	Bone§ F
Shawnee	2,261,240 (20)	4.07† ±2.39	11.55 ±3.58	43.87 ±7.73	34.39 ±5.97	21.69 ±5.55	4.88 ±2.21	52.42‡ ±8.08	7.77 ±2.6	10.75 ±3.84	3.34 ±2.01
Johnson	2,255,784 (11-17)	8.70 ±3.15	12.57 ±3.35	36.69 ±5.87	28.94 ±5.13	29.34 ±5.14	13.18 ±3.68	37.70 ±6.56	10.46 ±2.78	6.43 ±2.06	1.49 ±0.86
Wyandotte	1,783,759 (13-17)	21.01 ±5.56	15.13 ±4.29	36.82 ±6.73	23.68 ±5.03	26.18 ±6.31	8.84 ±2.95	38.68 ±7.45	14.5 ±3.75	6.43 ±2.06	1.49 ±0.86
Sedgwick	-0-	15.92 ±3.33	13.90 ±2.79	55.60 ±5.90	27.79 ±3.86	22.60 ±3.80	8.01 ±1.97	37.52 ±4.93	9.21 ±2.04	5.28 ±1.72	3.21 ±1.48
STATE	N/A	8.74 ±0.84	12.15 ±0.92	44.85 ±1.87	28.99 ±1.38	23.95 ±1.43	10.45 ±0.87	30.26 ±1.48	8.37 ±0.69	4.90 ±0.64	2.63 ±0.45

* > Sedgwick ($p < 0.05$)
† < Sedgwick ($p < 0.05$)
‡ > State ($p < 0.05$)
§ Johnson and Wyandotte county were analyzed together for bone cancer.

the Sedgwick County average, Wyandotte County is significantly elevated only for total cancer in males. In contrast, total cancer in males in Johnson County and liver cancer in males in Shawnee County are significantly lower than in Sedgwick County. There are no significantly high cancers for both males and females for fluoridated counties.

With regard to the pattern of cancer rates, the positive associations with fluoride are for female rectum, female pancreas, and male bone. However, for male rectum an inverse association holds for the fluoridated counties. For male pancreas there is no consistent relationship, either positive or negative. Sedgwick County has the highest rate for male pancreas. For male bone cancer there is a pattern of increasing rates with increase in person-years of exposure. However the same pattern does not hold for females. Female bone cancer is higher in Sedgwick County than in Wyandotte and Johnson counties combined. Considering the fluoridated counties only, there is an inverse association with fluoride for male stomach cancer, male rectal cancer, male and female liver cancer, and female bladder cancer.

Comparing Sedgwick County's rates with those of the other counties *(Table IV)*, it can be seen that rates for Sedgwick County exceed rates for Shawnee County nine of 18 times. For Johnson County, the Sedgwick County excess is ten of 18 and for Wyandotte County, the Sedgwick County excess is only four of 18. For males, Sedgwick County's rates exceed those of the other counties 12 of 27 times. For females, the similar comparative excess for Sedgwick County is 11 of 27 times. The only sites where Sedgwick rates do not exceed any of the other

TABLE IV
CANCER SITES ELEVATED IN SEDGWICK COUNTY IN COMPARISON TO OTHER COUNTIES

	Shawnee	*Johnson*	*Wyandotte*
Total			
WM	√	√	
WF		√	
Stomach			
WM	√		
WF		√	√
Colon			
WM		√	
WF		√	
Rectum			
WM	√	√	
WF			
Liver			
WM	√	√	
WF	√	√	
Pancreas			
WM	√	√	√
WF			√
Kidney			
WM	√		
WF	√		
Bladder			
WM			
WF	√		
Bone			
WM			
WF		√	√

WM — White male.
WF — White female.

counties are for female rectum, male bladder, and male bone cancers. Thus, the Kansas data do not support any hypothesis linking artificial fluoridation with cancer.

Conclusion

Studies done by the NHF attempting to link artificial fluoridation to cancer are invalid. They do not meet even minimally acceptable standards for generally accepted epidemiological principles. Additional studies done by several other scientific investigators indicate that fluoride is not even associated with cancer. Cancer mortality data presented for Kansas indicate no association of artificial fluoridation with cancer. This Kansas data included exposures of up to 20 years.

At this time, the relationship of cancer to artificially fluoridated water has been extensively studied, perhaps more intensively than many other chemicals in the environment. It must be concluded that current evidence does not support the hypothesis that fluoride is associated with cancer, much less causes it.

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Plasmapheresis

A Therapeutic Option for Treatment of Selected Patients

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The efficacy of plasmapheresis in a patient with mixed cryoglobulinemia and pancytopenia is shown. The cryoglobulin was shown to have precursor cell antibody activity, and its depletion by plasmapheresis resulted in improvement of blood counts. Indications, limitations, and guidelines for plasmapheresis in various diseases are discussed.

ESSENTIAL mixed cryoglobulinemia was defined, when first reported in 1964, as the syndrome of weakness, arthralgia, and purpura occurring in the presence of cryoprecipitable immunoglobulins.[1] As many as 50 per cent of patients will develop glomerulonephritis, which is considered a poor prognostic indicator. Other signs and symptoms include malaise, fatigue, hepatosplenomegaly, lymphadenopathy, and peripheral neuropathy. Plasmapheresis has been successfully used for the acute treatment of several autoimmune, vascular, cutaneous, renal, and neurologic diseases.[2-3]

Case Report

A 72-year-old white male had begun to complain of pain and swelling in hands, feet, ankles, and knees at the age of 26 years. This arthritis was non-deforming and continued in variable intensity thereafter. At the age of 40 years, he noted stinging purple patches on his lower extremities, particularly following cold exposure. At the age of 54 years he was found to have acute diffuse proliferative glomerulonephritis, complicated by modest hypertension (190/110) and congestive heart failure. Cryoglobulins were detected which were composed of polyclonal IgG with monoclonal IgM. Antinuclear antibody was negative; rheumatoid factor was 1:20 by latex test. Hepatitis B surface antigen and antibody were negative. He was treated with cyclophosphamide but leukopenia prompted discontinuance of the drug. When blood counts returned to normal, azathioprine was started, and the patient experienced deterioration of renal function during the next six years. When the patient was 60 years old, increased renal insufficiency (serum creatinine 2.5-3 mg/100 ml) was noted. Profound malaise, worsening congestive heart failure, and vasculitic skin ulcers were other major clinical problems. Pancytopenia (hemoglobin, 7.5; white blood cells, 1500; platelets, 100,000) with a hypocellular bone marrow necessitated discontinuance of azathioprine. Prednisone, 1 mg/kg in divided daily doses, was started, but blood counts did not improve. At this time, cryoglobulins, expressed as 7 per cent cryocrit, were present. Bone marrow culture results, reported below, demonstrated an *in vitro* precursor cell suppressive effect derived from the cryoglobulin. Plasmapheresis was undertaken, with cryocrit falling to 1.5 per cent after the first exchange, and to 0.5 per cent after the second and third exchanges. Renal function stabilized, and two weeks later, cell counts rose to 4500 WBC; hemoglobin, 9.5 gm/100 ml; and platelets, 210,000. He maintained adequate counts until his sudden death six weeks later following a cerebrovascular accident.

Evaluation of Granulocyte Precursors

The colony forming unit in culture (CFU-C) assay was performed as described earlier.[4, 5] Briefly, 2 x 10^5 bone marrow cells were plated on top of feeder layers into triplicate tissue culture plates (Falcon Plastics No. 3002, Oxnard, CA). The bone marrow cells were mixed in 0.5 ml of 0.6 per cent agar (Bacto agar, Difco Laboratories, Detroit, MI), 0.15 ml fetal calf serum (Kansas City Biologicals, Lenexa, KS). The feeder layer was made up of 1 ml of 0.5 per cent agar containing 1 x 10^6 fresh human blood mononuclear cells. The latter were prepared by the standard Ficoll-Hypaque centrifugation

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TABLE I
CRYOGLOBULIN ANTI-PRECURSOR CELL ACTIVITY

Bone Marrow Study Source	Manipulation	CFU-C* #
Patient	—	2
Control	—	47
Control	Patient's plasma	0
Control	Patient's plasma minus cryoglobulin	34
Control	Solubilized cryoglobulin	0

* Colony forming unit in culture.

TABLE II
PARTIAL REVERSAL OF PRECURSOR CELL ABNORMALITY BY PLASMAPHERESIS

Bone Marrow Study Source	Plasma Addition	CFU-C* #
Control	—	47
Control	Patient preplasmapheresis	2
Control	Patient one week post-plasmapheresis	37
Control	Patient four weeks post-plasmapheresis	32

* Colony forming unit in culture.

method. The plates were incubated at 37C in humidified atmosphere of 5 per cent CO_2 in air, and the cultures were scored on day seven for the presence of colonies made up of 40 or more cells.

Evaluation of plasma or cryoglobulin inhibitory activity was performed by incubating the bone marrow at 37C for 60 minutes with 100 μl of patient's or control's adsorbed plasma in the presence and absence of 100 μl of fresh guinea pig serum. Plasmas were adsorbed as described by us earlier to exclude anti-HLA, anti-T, anti-platelet, anti-granulocyte, and anti-red cell activity.[6] Proof that the inhibitory factor was the cryoglobulin was performed by adding purified cryoglobulin to the bone marrow cells prior to plating.

Results

As can be seen from *Table I*, the patient's bone marrow cells formed very few CFU-C as compared to the control marrow. Patient's plasma inhibited the control bone marrow CFU-C. The inhibitory plasma factor was partially removed upon depletion of the cryoglobulin. The solubilized cryoglobulin by itself was capable of inhibiting the control marrow *(Table I)*. Plasmapheresis resulted in removal of the inhibitory factor. The patient's post plasmapheresis plasma was not inhibitory to the control bone marrow *(Table II)*.

Discussion

Immune mechanisms in pancytopenia were shown by us to be heterogenous.[7] They could be due to hyperactive suppressor cells,[4] presence of antiprecursor cell antibody,[8] or abnormalities of bone marrow microenvironment.[7]

In this report we have conclusively demonstrated that the cryoglobulin of our patient with mixed cryoglobulinemia had precursor cell antibody activity. Depletion of the patient's plasma of cryoglobulin resulted in partial reversal of inhibitory activity. Addition of purified cryoglobulin resulted in inhibition. Plasmapheresis was effective in removing the inhibitory cryoglobulin. It seems that the patient's cryoglobulin is unique in having anti-precursor cell activity since cryoglobulin of three patients with collagen vascular disease who had normal peripheral blood counts (two with systemic lupus erythematosus, one with rheumatoid arthritis) and two other patients with mixed cryoglobulinemia failed to demonstrate anti-precursor cell activity (unpublished observations). The unique antibody activity of our patient's cryoglobulin is unclear at the present time.[9] Unique viral components within the cryoglobulin could be contributing factors. Viral infections were shown to induce pancytopenia.[10, 11]

Though plasmapheresis is still an experimental therapeutic procedure in several disease states, its value has been reported in Rh incompatibility, hyperviscosity syndrome, dysproteinemias, and homozygous familial hypercholesterolemia.[12] In selected cases in which excessive amounts of antibodies or immune complexes were shown to be involved in the disease process, plasmapheresis was shown to be beneficial.[2, 3] We have recently reported the beneficial effects of plasmapheresis in a patient with active systemic lupus erythematosus with pancytopenia in whom anti-DNA antibody was shown to have precursor cell antibody activity.[8] Similar beneficial effects were reported upon removal of glomerular basement membrane antibody in Goodpasture's syndrome,[2] of acetyl choline receptor antibodies in myasthenia gravis,[13] and of cryoglobulin in cryoglobulinemia.[14]

Several important points that should be emphasized in the use of plasmapheresis as determined by our experience and that of others[2, 3, 8, 12, 13] include: (1) plasmapheresis should be limited to patients who have life-threatening manifestations and in whom evidence of circulating factors or antibodies has been shown to be contributing to the disease process; (2) plasmapheresis should be followed by immunosuppressive drug therapy to avoid a rebound in humoral immune responses;[8, 13] (3) using plasmapheresis for the treatment of autoimmune-inflammatory disease should be preceded and followed by close monitoring of relevant antibodies or immune reactants so that a critical analysis of the efficacy of this procedure in humans can be clearly evaluated in the future; (4) plasmapheresis does not prevent an ongoing primary immune response[8] nor prevent eventual production of a primary de novo antibody response (we have reported earlier that weeks after plasmapheresis, and while on moderate doses of corticosteroids and cytotoxic drug therapy, a patient with systemic lupus erythematosus produced IgM class anti-DNA antibody); and (5) plasmapheresis could deplete a large load of antibodies and cryoglobulins and thus could reverse a pathologic process by making it more susceptible to the anti-inflammatory immunosuppressive effects of steroids and cytotoxic drugs.

Thus, for selected patients with active, life-threatening disease unresponsive to conventional therapy, plasmapheresis provides an acute albeit temporary experimental therapeutic option. Additional experience may show whether patients with less severe disease activity may become increasingly responsive to conventional treatment.

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Hyperprolactinemia

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Family Practice Challenge

The Social Development of Children With Chronic Illness

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CHRONICALLY ILL children may engage a significant proportion of a family physician's time. Advancements in medical technology and skills have resulted in a longer survival time for such children. The child's survival, however, is not without significant impact on his/her emotional growth and development. Personality changes varying from dependent, hysterical, withdrawn reactions, to aggressive behavior patterns marked by tendencies to deny illness[1-4] are common. Recognition of the impact chronic illness may have on the child's social development is an integral part of the family physician's role in the management of these children.

Case Report

A 7-year-old black female with a two year history of ulcerative colitis presented with abdominal pain, bloody diarrhea, and anemia. She was reported to have numerous school absences and limited contact with peers. By her own description, she was not disciplined as was her brother, nor was she expected to perform usual tasks for her age. Observation of the patient with her family confirmed this account. The patient successfully used physical symptoms to control her environment and the behavior of others toward her.

Physical findings included the following. The patient weighed 14.5 kg and was 114 cm tall, both below the third percentile. Her extremities were thin and her abdomen protuberant. Hemoglobin was 5.9/dl, and chest x-ray showed an enlarged heart. The diagnosis of ulcerative colitis was reconfirmed by barium enema and by biopsy of the sigmoid colon.

Following treatment with RBCs, iron, Azulfidine and Prednisone, the patient's somatic symptoms subsided. Although she improved physically, she continued to be isolated, withdrawn, and uncooperative. The patient was hospitalized for three months for evaluation and treatment of her ulcerative colitis as well as for evaluation and treatment of psychosocial problems. In addition to the family physician, the Family Practice social worker and the pediatric nursing staff were included in the planning team. Child Psychology was consulted, and when the patient's physical condition permitted, she was transferred to that service with continuing Family Practice followup.

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A child with chronic illness presents a formidable challenge to the family physician in the role of manager of the disease. Awareness of the negative impact of such illness on the child's social development and the potential for dysfunctional relationships in the family unit enables the family physician to serve as a stabilizer and facilitator. The family physician should strive to reach treatment goals and help the child and his/her family to achieve optimum functioning within the limits set by the chronic illness.

Definitions

Chronic illness is a disorder with a protracted course which can be progressive or fatal or associated with relatively normal life span but with impaired functioning. Long term disorders, as distinguished from non-life-threatening acute illness, have the potential to significantly and permanently interfere with physical and emotional development.[4, 5]

Social development refers to the way children behave with other children and adults as well as how they function in the social roles prescribed by families and communities.[6]

The interaction between the above concepts is important because normal social development depends upon multiple factors, including physical development, nurturing family relationships, intellectual development, and learning and social contacts in a variety of settings. Chronic illness has the potential to disturb this development.

By history and by observation of the child's behavior as an inpatient, it was obvious that illness had limited her activities, expectations, and social development far beyond physical impairment. She appeared frail and unwell, and those around her were reluctant to require much of her, including minimal socialization. This overreaction to the physical problem created another handicap for the child. Intellectually as well as socially this child lagged behind her peers. Her potential for a life with rewarding friendships and self-satisfaction would have been jeopardized if this situation had been allowed to persist.

Discussion

In the case of this patient, as well as those with other chronic disease states and their possible sequelae, the family physician can serve as a "normalizer" very early in the course of treatment. This case represents only one of a host of chronic diseases that can have a profound impact on the social development in general, and the autonomy development, in particular, of these children.

Social development refers to the way children behave with other children and adults, as well as how they function in the social roles prescribed by families and communities. Expectations of social behavior change with the age of the individual.[7] An autonomous person's self-concept is not a projection of others' opinions, but a self-image based on rewarding experiences in a variety of situations.

In the individual with a chronic illness, *i.e.* a disorder with a protracted course — which can be progressive or fatal or associated with relatively normal life span, but with impaired functioning — self-concept and learning can be affected profoundly by the illness or disability. The role of the family physician in keeping these interferences to a minimum can be aided by a knowledge of the child's environment and the parents' expectations and fears. The environment of the chronically ill child is not the same as that in which normal children learn to interact with others and to find their place in the world. The family physician should alert the parents to the fact that chronic disease creates a reality of its own. The nature of the disease process itself and the treatment procedures change the child's body, energy, and adaptability; attention is focused on lengthy treatment and procedures rather than on normal development challenges.[8] The impact of chronic illness on the family structure and relationships, the child's reaction to the stress of illness and hospitalization, and his stage of development affect social development, self-esteem, and ability to function in the larger environment.

The family physician should be aware that the parents — especially the mother — may be burdened by a sense of guilt for producing a defective child. The dependency of the sick child places a strain on the parents' marriage as well as on other children in the family who may resent the disproportionate time and effort devoted to him/her.[9] An early referral by the family physician to a person who has the time and training in marriage and family therapy may be beneficial.

It is well known that a family with adequate financial and emotional resources to meet the needs of the chronically ill child will be better able to offer support for a child facing life-threatening obstacles.[10]

The important role of the family physician in maintaining family confidence may help parents to reduce their anxiety. They should become, with the physician and other allied health professionals, part of a collaborative health maintenance network. Attention to comprehensive care should extend beyond the hospital or office to community support systems. The family physician should serve to continually remind all involved that the goal of treatment is to establish functioning at the *highest level possible* within the limits of the chronic illness.[11] These limits may be subject to change with medication, surgery, and emerging techniques.

Parents, as well as personnel working with children who have chronic diseases, need a thorough understanding of the normal progression of child growth and development. In addition to the need for treatment of his disorder, the patient has nonmedical needs specific to his stage of development. The family physician can be instrumental in helping parents to see developmental potential and to provide daily stimulation to realize these potentials. This reduces parents' fears and increases the child's capacity to make effective use of medical intervention. Social functioning and self-esteem need not be sacrificed to a chronic disability. The family physician's role in assisting the family and patient to realize this fact is an invaluable one.

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Chemical Wastes

Assessing Health Effects From Environmental Exposure

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CHEMICAL WASTES in the environment are now considered one of the major public health problems of the 1980s. The Love Canal disaster in upper New York State has alerted us to the consequences of decades of improper waste disposal. The site was used for the dumping of chemical and municipal waste for about 25 years, ending in 1953 when it was sold to the Niagara Falls board of education. A school was subsequently built on the 16-acre site, and hundreds of homes were built on and near the site. Subsequently, more than 80 buried chemicals have been identified there,[1] including benzene (a leukemogen) and seven other chemicals that are carcinogenic in animals. Currently, several hundred families have been evacuated, and it has been suggested that an increased risk of spontaneous abortions and congenital malformations exists. How can one assess the potential health risks associated with such an enormous uncontrolled chemical laboratory?

Health Effects in Those Exposed

When confronted with the presence of such a site, one immediate need is to ascertain or estimate the concentration and quantity of chemicals present. Unfortunately, adequate records of chemical contents are generally unavailable. Only recently has industry become concerned with this problem, largely because of pressure exerted by the Environmental Protection Agency (EPA) and others. Therefore, for most dumpsites, environmental (air, water, and soil) sampling is necessary. Such measurements are obviously affected by the topography and geology of the site as well as by rainfall, water table, surface drainage, wind speed, and wind direction. Chemicals buried inside containers may leak out in the future, but be unmeasured today. On-site and off-site concentration levels need to be monitored over time and place. Although direct measures of human exposure (*e.g.*, tissue, blood, and urine levels) are possible, these may not be necessary in the early stages of analysis unless there are health related complaints.

Recent illegal dumping of industrial wastes in Kansas City, Kansas has emphasized the need to alert Kansas physicians to potential health consequences to residents living near such dumpsites. The information given here is intended to increase the awareness of the physician to these potential health problems and the important role that the physician can play in the alerting process.

Another area of concern relates to the population exposed. Do the chemicals pose a risk to children playing on the dumpsite, to residents of the neighborhood, or to adults working nearby or involved in the operations or clean-up of the site? If off-site chemical migration occurs, will it enter private wells or public drinking water supplies? Will it leech into the basements of houses, be carried airborne to nearby residences, or enter the food chain? Personal characteristics such as age, sex, and occupation clearly are important variables in the human response to environmental agents.

Ultimately the problem becomes one of assessing human health effects, both qualitative and quantitative. Potential effects are estimated, based on previous animal and human studies. For example, if 2.4 kilograms of dioxin (2, 3, 7, 8 TCDD) are found in drums in a dumpsite, the potential acute effect is about 300,000 deaths total if the entire content were to be consumed. This effect is calculated using dose levels established experimentally in rabbits. This is one way a single site can be compared to others. Another health endpoint for dioxin might be liver cancer. Estimates can be made of the potential increase in liver cancer rates over background levels. In that case it might be helpful to have a background

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level of liver cancer in the population at risk for comparative purposes. Such data is readily available in Kansas for liver cancer mortality, but not for some other possible outcomes of dioxin exposure — such as chloracne, liver fibrosis, reduction in nerve conduction velocity, congenital malformations, and thymus atrophy.

In addition to the impact of a single chemical, the effect of multiple chemicals acting either additively or synergistically must be considered. This complicates the picture considerably. For example, polychlorinated biphenyls (PCBs) have been found in waste oils. These are believed to cause chloracne, mucous membrane irritation, fatigue, reproductive system damage, and possibly liver cancer.[2] The degree of synergistic or additive effect of PCBs and dioxin is undetermined. Another example involves carbon tetrachloride (CCl_4) which, along with dioxin, has been detected buried in the Love Canal site. The potential chronic effects of CCl_4 include toxic hepatitis, hematuria, and possibly liver cancer. Susceptibility to CCl_4's effects is enhanced by alcohol use.

It is not always necessary to rely on estimating potential health effects. By means of epidemiologic studies utilizing questionnaires, medical examinations and laboratory analyses, changes in health status in an exposed but presumably healthy human population can be assessed. Adequate followup is needed if diseases requiring long latency periods are to be monitored. Oncogenic agents might take 15, 20, or more years to produce their effect. On the other hand, teratogens and other agents affecting the reproductive system might have a noticeable impact within months. Another approach consists of investigating unusual clusters of disease (*e.g.*, type of disease, occupation, or residence) to ascertain what exposure conditions were common in the cases but rare in the controls. The careful selection of controls and the adequacy and objectivity of history taking can make or break this kind of study.

Kansas City Dumpsites

One example of an industrial waste problem in Kansas was brought to the attention of the EPA in February 1980. Industrial waste from a secondary aluminum smelter was disposed of in seven unauthorized locations in Kansas City, Kansas during 1979 or 1980. These locations were on vacant lots and in residential areas. The total estimated tonnage involved was approximately 57,100. Material from six of the seven sites was eventually moved to the Johnson County landfill for proper disposal. Laboratory analysis of the slag material for metals showed that at least ten metals were definitely present that (1) were in excess of what was normally expected in soil, and (2) had potential health effects that could be attributed to them. These metals were silver, arsenic, barium, beryllium, cadmium, chromium, iron, manganese, nickel, and lead. Of these ten, the five metals in greatest excess were arsenic, beryllium, chromium, nickel, and lead. Arsenic is a known human carcinogen for skin and lung. Hexavalent chromium is a presumed lung carcinogen, and nickel is a presumed lung, larynx, and nasal cavity carcinogen in humans. Beryllium is a possible lung carcinogen in humans. Possible non-carcinogenic chronic effects from exposure to those five inorganic metals include those shown in *Table I.*

TABLE I
HEALTH EFFECTS FROM EXPOSURE TO METALS DUMPED IN KANSAS CITY, KANSAS

Arsenic — skin and mucous membrane irritation, conjunctivitis, dermatitis, hoarseness, perforated nasal septum, cough, chest pain, dyspnea, throat constriction, keratoses, weight loss, and peripheral neuritis
Beryllium — contact dermatitis, conjunctivitis, cough, weight loss, dyspnea, pneumonitis, and bone and joint pain
Chromium — allergic dermatitis, coughing, wheezing, headache, dyspnea, pain on deep inspiration, fever, loss of weight, tracheobronchial irritation, lacrimation, nasal itch and soreness, perforated nasal septum, chronic asthmatic bronchitis, skin ulceration, and dental erosion
Nickel — skin sensitization and itching, conjunctivitis, irritated mucous membrane of upper respiratory tract
Lead — intestinal colic, inhibition of hemoglobin synthesis, wrist and foot drop, reduced nerve conduction velocity, dullness, irritability, ataxia, loss of memory, encephalopathy in children, miscarriages, stillbirths, teratospermia, and kidney tubule damage

In order to ascertain the health effects among those living near four of the sites, a questionnaire survey was conducted by the Kansas City, Kansas-Wyandotte County Health Department in cooperation with both the Kansas State Department of Health and Environment and the Centers for Disease Control, US Public Health Service, Atlanta, Georgia. This preliminary survey included interviews with 154 exposed and 40 non-exposed residents. Questions were asked about ten specific symptoms occurring both at the time of the survey and during the eight months preceding the removal of the waste

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Chronic Renal Failure

T. B. WIEGMANN, M.D.;* D. C. ARTZER, M.D. *and*
R. V. PATAK, M.D., *Kansas City*

Chronic renal failure (CRF) will be defined here as existing or evolving irreversible failure of renal function. This broad definition encompasses a wide spectrum of clinical disorders from specific renal diseases to renal complications of systemic illnesses. CRF may be the result of an indolent process prolonged over many years, and progression may be slow. CRF may also evolve more rapidly over weeks and months from an incident of acute renal failure with incomplete functional recovery. The term CRF is often used interchangeably with end stage renal disease (ESRD). This denotes final progression to complete renal failure and usually implies the need for dialysis or transplantation. Recognizing the presence of CRF is the initial task of the physician. Frequently, history and physical examination provide many clues, although de novo recognition results most frequently from the inspection of laboratory values obtained during office or hospital evaluation.

Diagnosis

After recognition, the physician must establish whether renal failure is acute or chronic and determine the rate of progression. History and physical examination are of value in providing important signals. For example, it is important to seek evidence of familial or hereditary renal conditions or for systemic medical diseases such as diabetes mellitus. Previous examinations may document hypertension or abnormal urinary findings such as proteinuria or hematuria. Drug history may prove extremely informative since numerous drugs, including antibiotics, contrast-media examinations or anti-inflammatory drugs, may cause renal damage. Possible exposure to heavy metals, toxins, or organic solvents should also be pursued. Finally, almost every major medical illness provides an important background for the development of CRF *(Table I)*.

The duration of presenting symptoms may help define time of onset of renal dysfunction. Metabolic dysfunction may present as anorexia and weight loss whereas peripheral neuropathy may result in paresthesia and pruritus. An important degree of anemia may be signalled by pallor and exercise intolerance. Cardiac failure, fluid retention, and possible pericarditis should be suspected when dyspnea, peripheral edema, nocturia, or chest pain are found. Many other symptoms or signs may be variably present *(Table II)*. Finally, measurements of serum creatinine and urea nitrogen will easily confirm an initial diagnostic suspicion. Chronic rather than acute renal dysfunction may be supported by a finding of anemia, hyperphosphatemia, and hypocalcemia. Similarly, broad "waxy" urinary casts are found in many instances of advanced chronic disease; however, an essentially normal urinalysis does not preclude the existence of CRF.

The laboratory evaluation of CRF proceeds as suggested for evaluation of acute renal failure. A plain x-ray film of the abdomen is essential to determine size, shape, and kidney position as well as

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TABLE I
COMMON HISTORICAL CLUES TO THE PRESENCE OF CHRONIC RENAL FAILURE

1. Family history of	Diabetes mellitus Hypertension Cardiovascular disease Renal diseases including polycystic renal disease
2. Recurrent urinary tract infections	
3. Obstructive events including	Renal colic Prostatism Renal stones Prolonged childhood enuresis
4. Episodes of acute renal failure acute glomerular disease	
5. Multiple system disease	
6. Chronic drug intake including	Analgesics Non-steroidal anti-inflammatory drugs

TABLE II
SIGNS AND SYMPTOMS OF ADVANCED CHRONIC RENAL FAILURE

1. General	Lack of energy Mental changes Somnolence Stunted growth (children) Hypothermia Anorexia, nausea Weight loss
2. Neurologic	Peripheral neuropathy Muscle weakness Asterixis Paresthesias Pruritus Hiccups
3. Cardiovascular — Respiratory	Pronounced ventilation due to acidosis Hypertension Nocturia Pericardial friction rub Congestive heart failure Pulmonary infiltrates
4. Extremities	Bone/joint pain Spontaneous fractures Edema
5. Skin	Excoriations Sallow to yellowish-brown complexion Petechiae Ecchymosis Epistaxis
6. Eyes	Blurred vision Conjunctival/corneal calcification and irritation Fundal hemorrhage and exudation Arteriolar changes

presence or absence of renal stones or calcifications. Where available, sonography and isotopic scanning are recommended to look for evidence of obstruction and symmetric or asymmetric changes in renal blood flow and function. Bilaterally small kidneys with delayed symmetric uptake and excretion of I^{131} hippuran or its analogues afford good evidence for a chronic process. General use of intravenous pyelography (IVP) is no longer recommended because renal function may deteriorate further with radiographic contrast examinations such as IVP and contrast enhanced computerized tomography (CAT-scanning). Particular caution must be exercised in patients with dehydration and with larger doses of contrast (*e.g.*, infusion IVP) and in those CRF patients with high grade proteinuria as in diabetic renal failure. Arteriography is rarely of diagnostic value with advanced symmetric dysfunction. Renal biopsy will usually not be considered once the kidneys have become small or when a specific systemic condition is present and known to cause CRF (*e.g.*, diabetes mellitus). When renal failure evolves rapidly during a period of weeks or months, or when the cause remains undefined, early arrangements should be made for renal biopsy.

Reversible Elements

The most complete evidence of CRF must not prevent the diligent search for a reversible cause or component and superimposed acute conditions. Any cause for acute renal failure may be superimposed on existing chronic changes. Even minor acute events may cause significant dysfunction and additional damage in the presence of pre-existing disease. Treatment of lesser acute complications often may not result in the expected resolution, particularly if

the condition is well advanced. Nonetheless, if a reversible condition is treated aggressively in the presence of underlying CRF, partial reversal may obviate the need for chronic dialysis. There is little precise information about the natural history of advanced chronic renal failure, particularly the true incidence of reversible components or acute complications in patients with end-stage renal disease. One study by Maher and associates found improved renal function after treatment for coexisting complications in 20 per cent of patients with end stage renal disease. Importantly, these improvements persisted for an average of more than a year. This emphasizes the importance of considering acute components in each case.

The most common reversible conditions or acute complications are hypertension, urinary tract infection, dehydration, primary cardiac decompensation, metabolic diseases, stone disease, and obstruction. Recent surgery and evidence for nephrotoxic drugs or contrast media investigations should also be considered. Most of these conditions are readily recognized and treated, frequently with significant improvement in renal function. Acute peritoneal dialysis may be used to advantage in advanced cases, and can provide the necessary time for additional investigations. Obstruction is particularly common and should be ruled out in every patient, especially elderly men. If obstruction is suggested by sonography or isotopic scanning, retrograde pyelography should be considered rather than IVP for additional evaluation of the urinary tract.

Prognosis

The prognosis of CRF in individual patients is influenced by numerous considerations predominantly related to the underlying disease and its intrinsic progression. The occurrence of acute complications may accelerate underlying disease by leaving residual damage despite aggressive therapy. It is therefore difficult to make long term predictions. The issue of progression must be addressed as early as possible using historical, clinical, radiological, and serial laboratory data. Rising serum creatinine concentrations reflect progressive deterioration in the endogenous clearance of creatinine, in turn signalling the continuous decrease in the rate of glomerular filtration and thereby renal function. A doubling in the serum creatinine concentration is equivalent to the loss of 50 per cent of nephron function. This change must be understood as a functional loss and does not necessarily reflect a similar decrease in the number of functioning nephrons.

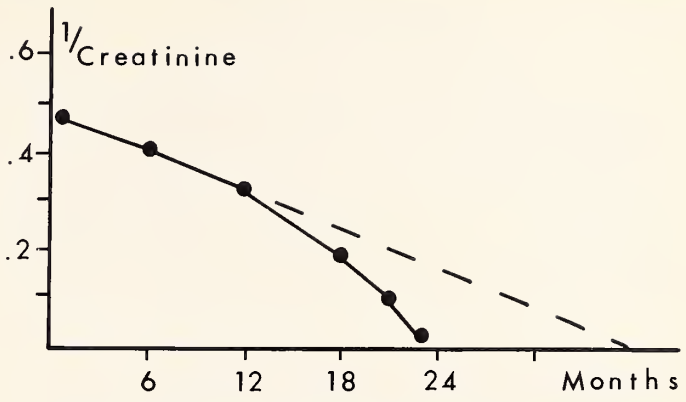


Figure 1. Progressive deterioration of renal function with time in a 59-year-old man with adult onset diabetes mellitus and hypertension. Initial 1/creatinine values are extrapolated (dashed line) to indicate projected time course to complete renal failure. Rate of progression accelerated after 12 months, prompting a search for acute complications.

If renal failure progresses indeed at a constant rate then serum creatinine concentrations will also increase steadily. At the same time, the reciprocal (1/creatinine) decreases at a steady rate. This value can easily be plotted on the patient's chart and provides a convenient way to illustrate the progressive deterioration of renal function with time. A minimum of three, preferably four, timed creatinine concentration measurements should be used, separated by at least several months. Frequently one can thus predict the time of complete renal failure or suggest the presence of acute problems. This is illustrated in *Figure 1*, which depicts a 59-year-old patient with adult onset diabetes mellitus and hypertension. Evaluation of 1/creatinine values showed a steady decline during a period of 12 months. The fourth visit suggested some possible acceleration in his disease process, and followup periods were shortened. The next visit confirmed a distinct change in the rate of progress compared with the earlier predictions based on the first three measurements. Such obvious change prompted a search for acute complications because treatment of complications will frequently slow recent acceleration. In this instance, antihypertensive therapy was intensified in the absence of other complicating factors. Nonetheless, the patient failed to respond and required hemodialysis two months later.

Initiating Treatment

Early decisions about dialysis are necessary in patients with advanced conditions that fail to re-

TABLE III
SELECTED ABNORMALITIES AND CORRECTIVE MEASURES IN CHRONIC RENAL FAILURE

Feature	*Indication*	*Management*
Azotemia	Creatinine clearance < 40 ml/min	Dietary management 1.0-1.25 gm protein/Kg/day 60% high biological value 60 meq Na/day 40-60 meq K/day
Acidosis	HCO_3 < 18 meq/L	$NaHCO_3$ as required
Hyperphosphatemia	PO_4 > 5 mg/dl	Phosphate binders with meals as necessary
Hypocalcemia	Ca < 8.5 mg/dl	Ca (carbonate), 0.5-2.0 g/d $1,25(OH)_2$ vitamin D only with proper PO_4 control
Anemia	Hct < 30%	$FeSO_4$ Folic acid
	Hct < 17% or patient is symptomatic	Transfusion

spond to measures for correction of acute problems. Similar urgency is also advised for patients with quickly evolving renal failure perhaps due to a rapidly progressing glomerular nephritis. In either case, rapid referral is the rule. Difficulties arise when, as is frequently the case, the disease follows an indolent course. The physician must then provide long-term aggressive management of underlying conditions (*i.e.*, hypertension, diabetes mellitus) or intervening complications. Active medical management is also required for a number of metabolic changes that arise with declining renal function, such as hyperphosphatemia. Other common examples and corrective measures are summarized in *Table III*. Once dialysis has been considered, laboratory values are used to determine the timing. Most nephrologists agree dialysis should begin when the creatinine clearance is at 5 ml/min or less. Earlier dialysis (9 ml/min) should be considered in patients with diabetic renal failure. Consideration of laboratory indications for dialysis must further be influenced by the severity of clinical signs and symptoms. This is necessary because individual patients may show varying degrees of tolerance in the face of advancing uremia. Dialysis must be started if anorexia, weakness, orthopnea, and other complaints functionally restrict life style. The advent of peripheral neuropathy, pericarditis, or generalized wasting heralds the urgent need for active therapy. Arrangements for long term therapy should be made early enough to prevent the appearance of such definite signs of clinical deteriorations.

Summary

Renal failure will usually be discovered from laboratory data and established as chronic renal failure by consideration of historical data, physical findings, and a few additional laboratory findings. The advent of permanent renal failure results in an incredible burden for the patient and family, and brings important responsibilities for the treating physician. Judgment of the length of remaining time is useful in making long term treatment decisions and in providing proper counseling. A reasonable estimate for prognosis and rate of progression should also be sought to determine eventual therapy as early as possible. Every effort should be made to define and aggressively treat acute complications and reversible components. Aggressive medical management of underlying conditions and expected metabolic complications is necessary to delay the advent of end stage disease and dialysis.

Self-Assessment Questions

1. The most common reversible element in chronic renal failure is:
 a. Infection
 b. Systemic disease
 c. Obstruction
 d. Hypertension
2. Renal biopsy is of value in the diagnosis of:
 Acute glomerulonephritis (T or F)
 Chronic renal failure (T or F)

3. A safe radiographic approach to chronic renal failure includes:
 a. Plain x-rays of the abdomen
 b. IVP
 c. Sonography
 d. Radioisotope scanning
 e. Retrograde pyelography
4. Choose the right statement(s) about CRF
 a. CRF is occasionally reversible
 b. CRF is frequently reversible
 c. CRF is not reversible
 d. CRF has frequently a reversible component
 e. None of the above
5. Calcium replacement therapy should begin first with vitamin D. (T or F)

(Answers on page 156)

Ovarian Cancer

(Continued from page 129)

In summary, recent changes in chemotherapy have made ovarian cancer a manageable disease. This is not a report of our data, but emphasizes that with proper staging, debulking and combination chemotherapy, survival is increased and cures are frequently obtainable.

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Chemical Wastes

(Continued from page 146)

material. The results showed that those living near the dump sites complained of an increased incidence of skin rashes (13.6%), cough (22.3%), eye and throat irritation (15.3-17.5%), and tiredness (21.4%). The stated symptoms were certainly consistent with the potential health effects that could be caused by these metals.

All of the symptoms were considered to be only of a mild or irritating nature, since very few individuals sought medical attention and no one missed work or school because of them. Detailed medical data on all of these individuals was not previously available, and this survey was not designed to gather such data. However, urine samples were collected to look for the presence of arsenic or lead as indicators of significant absorption of this waste material. All urine samples were negative for both metals. After the waste material was removed, the stated effects apparently declined as no further complaints were made to the health department.

Conclusion

The chemical waste problem is of vast magnitude. One example of this problem in Kansas has been cited. However, there are tens of thousands of chemicals in the marketplace and thousands of new ones introduced each year. Waste chemicals may be found throughout the environment and not merely in waste dumps. There is a need for suitable processes to recycle or completely destroy such wastes, not merely bury them. Should chemical exposures be suspected, or unusual clusters of disease found, the physician needs to notify the appropriate state, county, or local health officer. In this instance, as in the case of reportable infectious diseases, the physician functions as part of an early warning system. Such timely warning may assist in establishing epidemiologic studies, improve the chances for early detection of disease, and reduce exposure to the agent.

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Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Cimetidine

Question: I have heard that cimetidine (Tagamet) may be involved in drug interactions with several drugs metabolized by the liver. Which drugs are involved in these interactions?

Answer: Cimetidine, a histamine H_2 receptor antagonist is widely used in the treatment of peptic ulcers and other gastric hyperacidity states. Initial clinical experience suggested that this drug was relatively free of adverse reactions. Recently, however, reports of potential drug interactions with cimetidine have surfaced.

During 1979, several investigators reported on the potentiation of the hypoprothrombinemic effect of warfarin by cimetidine.[1-3] In these studies, it was shown that the metabolic clearance of warfarin was inhibited by therapeutic doses of cimetidine.

More recently, investigators have also found that the metabolic clearance of diazepam[4, 5] and chlordiazepoxide[6] was reduced with concommitant administration of cimetidine.

Klotz *et al.*[4] first reported on a possible interaction between cimetidine and diazepam in four healthy subjects. They found that even short term cimetidine treatment could significantly alter the half-life of diazepam (29.7 hours before treatment to 71.1 hours with cimetidine). The same authors later studied the clearance of diazepam after its administration to six healthy volunteers.[5] The half-life again increased significantly from a baseline of 33.5 hours before cimetidine to 51.3 hours after cimetidine. Based on this information, it might be expected that a potentiation of sedative effects would occur, and in fact five of six subjects experienced a much stronger sedation when both medications were administered.

The prolonged half-life of diazepam suggests that elimination of other benzodiazepine drugs might also be impaired. This suggestion was confirmed by Desmond *et al.*[6] who found chlordiazepoxide (Librium) clearance to be reduced twofold. The half-life of chlordiazepoxide increased from ten to 23 hours after pretreatment with cimetidine for one week.

The mechanism of this interaction is thought to be cimetidine inhibition of the cytochrome P-450-dependent metabolic pathway in the liver (involving n-dealkylation and hydroxylation steps).[7] It should be noted that two other benzodiazepines — lorazepam (Ativan) and oxazepam (Serax) — undergo glucuronidation only and are not metabolized by the cytochrome P-450 system. Patwardhan *et al.*[8] found the half-life and clearance of these agents to remain the same after administration of cimetidine, suggesting that the glucuronidation step is not influenced by cimetidine.

From these reports it appears that the interaction between cimetidine and benzodiazepines (diazepam, chlordiazepoxide) may be important in elderly patients and in those with liver disease, who may be more susceptible to the sedative effects of these drugs. The data also suggest that oxazepam and lorazepam elimination is unchanged, and thus possibly the benxodiazepines of choice in patients on concurrent cimetidine therapy. The cimetidine package insert has recently been revised to contain this new information on cimetidine interactions with warfarin and benzodiazepines.[9]

From emerging reports it appears cimetidine may

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The President's Message



The nursing shortage is not just the hospital's problem; it is our problem as well. We physicians rely on quality nursing to deliver quality health care. Many times nurses' actions and observations make the difference between life and death. We all know this. Nurses provide for our patients' needs 24 hours a day, seven days a week, whether in ICUs, CCUs, or on the medical/surgical floors. We, on the other hand, go in, direct and prescribe, leave, and for some time (at least physically) walk away from a particular problem.

We cannot alleviate all the dissatisfaction voiced by nurses (evenings, nights, weekends, for example), but there are some things we can do to improve morale and working conditions, so let us be about doing what we can.

We can give them greater recognition as essential professionals and an integral complement to medical practice. We must learn to talk *with* them and not *down to* them. We have no need to feel threatened or irritated by nurses' suggestions about patient care. A more cooperative approach will obviously lead to better job satisfaction and improved basic patient management.

A review of salaries — the tangible proof of recognition in our society — indicates that nurses receive inadequate compensation for their services. Although intensity of hospital care has increased dramatically in recent years, requiring greater clinical competence on the part of nurses, their remuneration has risen only modestly. They have taken the brunt of rate setting and cost-containment efforts. A nurse aid's income today is 71 per cent of a registered nurse's salary; a licensed practical nurse earns 76 per cent of a registered nurse's salary. Nurses' salaries do not compare well with those of other professions, including other health professions, such as physical therapists and laboratory technicians.

Constructive input and support from physicians can help this situation.

Fraternally,

Herman W. Hesterman M.D.

President



Double, Double, Toil and Trouble

It may come as a surprise in some sectors where the roles are being played out to the mutual satisfaction of both participants, but it is reliably and not infrequently reported that the ancient and honorable relationship between physicians and nurses is in trouble. This has been reported in a number of studies of varying statistical and professional validity, all better founded perhaps than these informal observations, but at least these come direct from a recognizable source without filtration through ideologic purpose, propagandized intent — or prior publication.

We acknowledge immediately that we are relying on the male physician-female nurse stereotypes which is not to ignore the increasing incidence of female physicians or male nurses but to recognize the traditional origin of this arrangement which puts a peculiar character on the conflict. There is no relationship —setting aside the patient for the moment — more closely associated or intimately identified with medical service than that of nurse and physician — and no comparable counterparts in other disciplines.

The current situation encompasses a number of features that have been frequently recorded in both professional (primarily nursing, to be sure) and lay periodicals (always fascinated by domestic squabbles). Nurses, the theme goes, are angry because they are forced to accept responsibility without equivalent authority, work at what they consider totally inadequate remuneration compared with that of physicians, and tolerate the abuse of physicians without rebuttal (no small factor, it is reported, in the shortage of nurses).

The traditional roles of physician and nurse originated with an accepted division of essential services. The physician, presumably because of more extensive knowledge and experience as well as that peculiar entity known as the physician-patient relationship, assumed the position of primary responsibility. The nurse, with what has been deemed an adequate academic base (much of it provided formally or informally by the physician — emphasizing the subservient position) and fulfilling the physician's orders, has applied her expertise with compassion and patient-interest as significant components along with a more constant attendance. In releasing the physician to cover a wider territory, her role has emphasized a supportive function to both patient and physician, filling an emotional need as well as providing professional service in a manner the physician could not, even as he directed the course of action. Hence the "angel" concept which is a natural outgrowth of the maternal source of care which is the real basis in its original and social context.

The physician-nurse team, productive as it has been, has carried the seeds of its own dysfunction. As medical care has grown more extensive and complex, the physician's responsibilities have grown more extensive and complex — and problematic — visiting upon the nurse additional responsibilities not necessarily of her choosing and beyond her accustomed functions. The litigious character of the age is not the only factor, but it exemplifies the case well. It is a common legal ploy to include in a suit, directed primarily against the physician, anyone within range of the alleged misservice. The nurse finds herself at risk — assaulted not only by the inherent distresses of such actions but by the seeming injustice of being held responsible for conditions over which she may have had no control. And this event, traumatic as it is, brings into focus other features of inequity and discordance that lurk in the background. The physician's single responsibility — to the patient — seems paltry to the frustrated nurse who must accept responsibility to the patient and to the physician as well (not to mention to her hospital-employer).

The pressure on the nurse to enhance her role by increasing her academic preparation has long been at work, and nursing education has become a large and important segment of the medical effort. It hasn't,

however, been an unalloyed success in providing equivalent status or in resolving the inherent conflicts. Even within the nursing profession, it precipitated contention between the "diploma" schools — generally associated with community hospitals — and those offering the baccalaureate degree. It was logical that expansion of nursing education toward a degree would be implemented in academic institutions equipped to provide the necessary curriculum. The nurse distinguished by a degree, however, was "elevated" to the administrative level of service, a situation which not only did nothing for bedside nursing, but enhanced the administrative structure in the time-honored principles of bureaucracy that pervades all disciplines. Physicians — particularly those in community hospitals — had mixed feelings, acknowledging the need for greater training for nurses, but deploring the lack of bedside service. And more than one nurse regretted her detachment from closer patient care even as the system prevented her from resolving the dilemma. The result, of course, was the upgrading of the aide to the role of licensed practical nurse to whom falls many of the contacts and functions relinquished by the nurse, now preoccupied with her paperwork and system-organizing.

This is not to ignore the increased utilization of the nurse in more specialized clinical services — anesthesia, obstetrics, and surgery in particular — and now the appearance, both inside and outside of the hospital, of the nurse-practitioner. This most recent arrival has, like other manifestations of extended service as they move into newly-opened territory, met with mixed reviews — satisfaction by those physicians who have utilized their services well, resentment by those who saw a threat to medical principles or to their positions. It is really a formalization of a situation that has long been growing as the individual physician has increasingly turned over to an astute and willing assistant as many duties as law, conscience, and patient acceptance would allow. But this phenomenon remains largely of individual application and is not likely to become so common as to have any deep impact on the basic alterations that are going on between the principals.

We are assured, too, that one of the prime annoyances in the traditional set-up is the inequity of remuneration. Nurses resent their subservient roles not simply because of the character of their responsibilities but because theirs are seemingly deemed less worthy of material gain than the physician's. This finds easy acceptance with the public, not only because its traditional sympathy lies with the underdog, but also because it derives a certain enjoyment from the discomfiture visited on physicians, who are known to be uniformly rich because of their exorbitant fees. It seems a singularly difficult point to counter to anyone's satisfaction. Should physicians reduce their fees or divide them (presumably in some legitimate way) with nurses? Should they go on some sort of hourly basis of service — and remuneration — comparable to that of nurses as a means of limiting their incomes (although, of course, requiring the acquisition of a large number of these economy types in order to get the work done)? One suspects that, although a healthy pay boost would certainly be acceptable to nurses, the complaint is more a reflection of their discontent with the general features of their function than a desire to bring physicians to a more modest level of financial reward. The more definitive character of the physician's service and more extensive responsibility which applies to all procedures and personnel involved, as well as the existence of that responsibility at all times regardless of his physical presence, may seem to the physician to justify his charges. But the nurse, feeling her own service and responsibility to be very little less, remains unconvinced and resentful.

This discontent is probably closely related to that other area of complaint so frequently cited — the abusive, at least derogatory, attitude toward nurses generally attributed to physicians. There is obviously no established professional policy to derogate nurses and it becomes a matter of personal behavior and not particularly amenable to organizational prohibition in the interest of good will. There is no doubt that the stresses of professional function may be vented on the handiest target, usually the nurse. This may be understandable to a degree, although obviously immature and indefensible; and understandability does nothing to make it agreeable to the nurse-target — even less when she has been put in the vulnerable position by the inequitable division of responsibilities. Everyone is acquainted with the petty tyrants who, by their own insecurities or sadism or whatever, make life miserable for those under their professional control — again most frequently the nurse. It is suggested, however, that these cases are relatively infrequent — certainly not typical of the profession — and the fact that they are frequently included in the nurse's bill of particulars against the physician stems more from the continuing subservience of her role in which she more and more visualizes herself as an equal without the authority to support it. It is more often than not an interpersonal relationship in which the nurse's real concern is in being denied by tradition and ground rules the right to indulge in a little catharsis of her own on this

personalized form of her frustrations and resentment. In the absence of any other solution, we offer the suggestion of a regular period of official dispensation when she will be permitted, without fear of retribution, to vent her spleen upon the physician who will be required, on pain of excommunication, to sit and take it.

This could have the salutary effect of giving the physician a more realistic view of himself without having to resort to the ministrations of his psychiatric colleague. And even those not given to such behavior are prone to forget that they have no secrets from nurses. Preoccupied as they usually are in their day-in-and-day-out contacts, they are prone to forget that in innumerable ways they expose their failures and foibles and that nurses, in this enforced and continuing scrutiny, come to know things about their characters and abilities that they don't know themselves — or admit. They are prone to forget the extent to which their exalted images depend upon the nurse's support for sustenance — and that any physician who has been in practice longer than 15 minutes is indebted to some nurse for some invaluable service — and education.

We suggest, therefore, that the problem comes down to these relative responsibilities — their character, their substance, their purpose, and their application in service to the patient. The disparities have been brought into relief by, as much as anything, the social and economic pressures of the day. The principals must have a healthy understanding of their individual roles and a dedication to their basic purpose — patient care — which acknowledges the interdependence that makes it all possible. The relationship requires judgment, understanding, and mutual respect on the part of both, but it is far too important an ingredient in the purpose to be sacrificed on the altar of ego or organization. — *D.E.G.*

Chronic Renal Failure

(Continued from page 151)

Answers

1. c
2. *True, False*
3. a, c, d, e
4. c, d
5. *False*

Suggested Readings

1. Depner, T. A. and Gulyassy, P. F.: Chronic renal failure. *Strauss and Welt's Diseases of the Kidney,* 3rd edition (Earley, L. E. and Gottschalk, C. S., eds.). Boston, Little, Brown and Company, Chapter 6, pp. 211-262, 1979.

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First Honors Convocation Address

(Continued from page 110)

setting of highly selected intellects, it is all the more difficult to distinguish oneself. Thus the accomplishment of those about to be honored is all the more significant. Today's recognition probably is more important than the student honorees can know at this time. However slightly, these recipients have already moved up a step in a crowd of general excellence. Though there will be exceptions, it is likely that a lifelong process is beginning here today, for there is certainly a measure of truth in Martin Fischer's observation that "There are no dark horses. Every great man in medicine was known as a yearling."[6]

So for students as well as teachers, today's celebration is an exercise in excellence. We should put aside our democratic qualms and literally revel in the elitism of genuine accomplishment. The recognition you are about to receive, in my estimate, is as valid and significant as any that will come your way.

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Drug Information

(Continued from page 152)

inhibit the metabolism of a wide range of drugs, as decreases in the metabolism of theophylline, propranolol, and phenytoin are now being reported.[10-13]

It appears prudent to closely monitor patients who receive cimetidine concurrently with other drugs metabolized by the liver. Interactions are likely to occur because of the high usage of these medications.

Submitted by
Joyce Generali, R.Ph.

References available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

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INDICATIONS: *Therapeutically* (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: • infected burns, skin grafts, surgical incisions, otitis externa • primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia) • secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis) • traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically*, the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: Not for use in the eyes or in the external ear canal if the eardrum is perforated. This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

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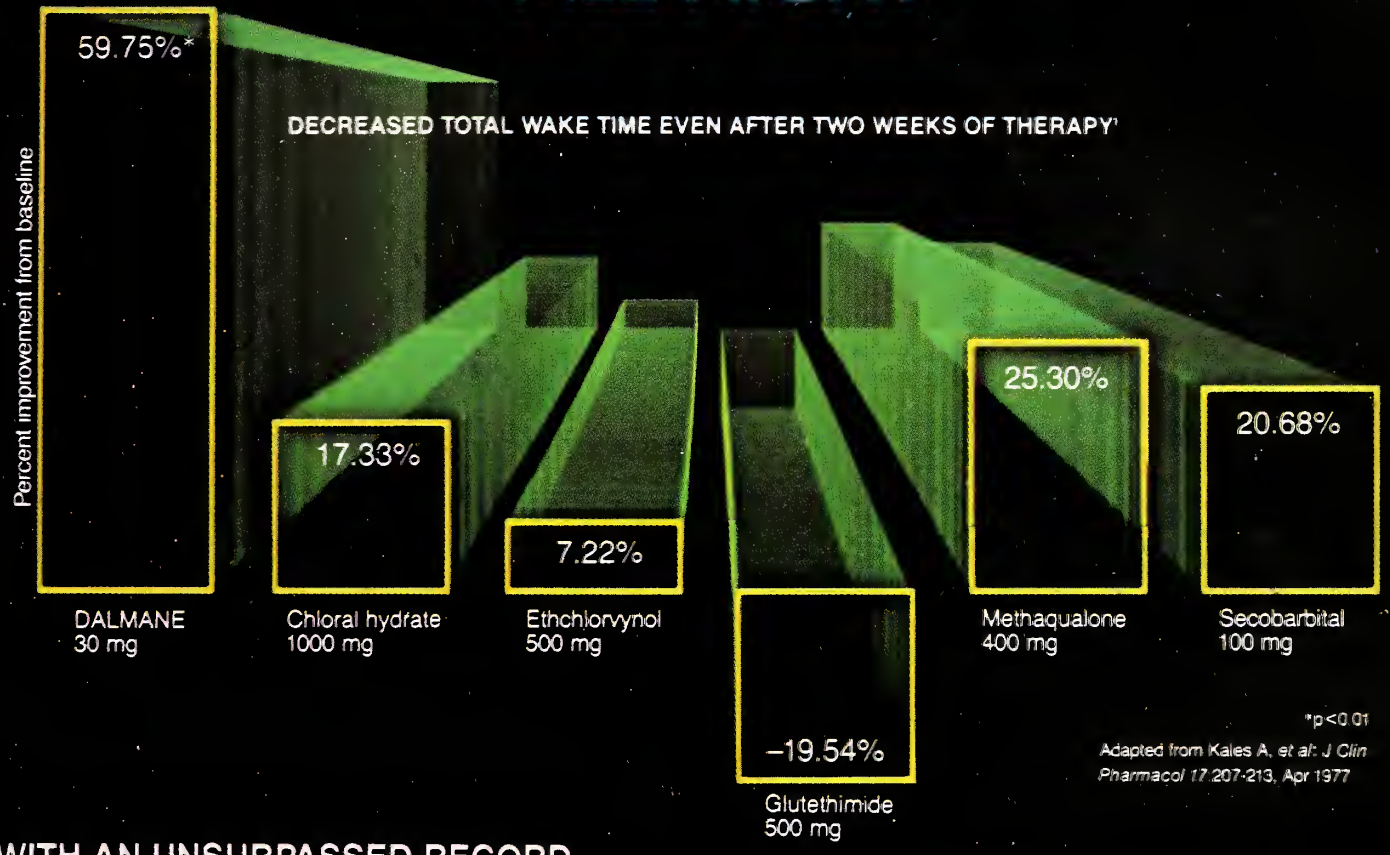
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–which have anticholinergic effects[11]

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–whose side effects may be troublesome for nonpsychotic patients[12]
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Dalmane does not cause significant worsening of sleep beyond baseline levels upon discontinuation.[4]

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Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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Family Practice Challenge

(Continued from page 144)

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2. Drinking to handle problems or relieve symptoms.



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4. Surreptitious drinking or gulping of drinks.
5. Tendency toward making alibis and weak excuses for drinking.
6. Refusal to concede what is obviously excessive consumption and expressing annoyance when the subject is mentioned.
7. Frequent absenteeism from the job, especially following weekends and holidays.
8. Repeated changes in jobs, particularly if to successively lower levels, or employment in a capacity beneath ability, education and background.
9. Shabby appearance, poor hygiene, and behavior and social adjustment inconsistent with previous levels or expectations.
10. Persistent vague physical complaints without apparent cause, particularly insomnia, stomach upsets, headaches, loss of appetite.
11. Multiple contacts with the health care system with disorders that are alcohol caused or related.
12. Persistent marital and family problems, perhaps with multiple marriages.
13. History of arrests for drunkenness or drunken driving.

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*Reference: 1981-82 *American Druggist Blue Book*

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INDICATIONS AND USAGE SU-TON contains pentylenetetrazol which may be helpful in the older patient as an analeptic agent when mental confusion and memory defects are present. SU-TON also contains vitamins, trace minerals, and iron, for those patients who may benefit by preventing the development of a deficiency.

CONTRAINDICATIONS Epilepsy, convulsive disorders or known history of sensitivity to any of the listed active ingredients.

WARNINGS The safety of this preparation during pregnancy and lactation has not been established. Use of this drug requires that the physician evaluate the potential benefits of the drug against any possible hazard to the mother and child.

PRECAUTIONS Although there are no absolute contraindications to pentylenetetrazol, it should be used with caution in epileptic patients or those known to have a low convulsive threshold or a focal brain lesion. Caution should be exercised when treating patients with high doses of SU-TON who have heart disease. While pentylenetetrazol does not act directly on the myocardium; the results from central vagal stimulation could cause bradycardia.

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OVERDOSAGE Signs and symptoms of acute overdose may be due principally from overstimulation of the central nervous system and from excessive vasodilatation with resulting autonomic nervous system imbalance. The symptoms may include the following: vomiting, agitation, tremors, hyperreflexia, sweating, confusion, hallucinations, headache, hyperpyrexia, tachycardia. Treatment consists of appropriate supportive measures. If signs and symptoms are not too severe and the patient is conscious, gastric evacuation may be accomplished by induction of emesis or gastric lavage. Intensive care must be provided to maintain adequate circulation and respiratory exchange.

DOSAGE AND ADMINISTRATION One tablespoonful (15 ml) 3 times a day 20–30 minutes before meals. This drug is not for use in children under 12 years of age.

HOW SUPPLIED Bottles of 473 ml (16 fl. oz.) NDC 0524-1015-16

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Bohan Lecture

(Continued from page 114)

parently frequent "Jimmy's Jigger," just off campus.

Dr. Russell Haden, the first Bohan lecturer in 1947, also came from Johns Hopkins. He came here as Professor of Experimental Medicine. During that time he formed a tremendous alliance with Dr. Thomas Orr, and together they pioneered in studies of fluid and electrolyte balance in surgical patients. From Dr. Haden I learned: "Surgeons *can* work with Internists."

Finally, I would like to pay tribute to my own personal hero in Kansas, Dr. Thomas Grover Orr. He came here in 1915 and took over the teaching of bacteriology from Dr. Major, and soon became the Chairman of the Department of Surgery. As a young man, Dr. Orr demonstrated compassion and good judgment and became the leading surgeon of the area. He entered World War I and became an authority on amputations. He also wrote a definitive textbook on general surgery. When I came here in 1949 he had reached the pinnacle of recognition as President of the American Surgical Association. He was also a founding member of the American Board of Surgery. An analysis of his bibliography, which I once did in preparation for an Orr lecturership, shows that he was one of the first clinical investigators in this country. From him I learned: "Patients are the ultimate source of information — study them, and *then* operate them." His bibliography also contained papers of a philosophic nature, including a treatise on the two available careers for students — family practice or specialization. One of his best speeches was given as President of the Southwest Surgical Congress. It was entitled "Doctors making Speeches." From that publication I should have learned that "Surgeons should avoid making speeches."

A recent aerial view of our medical school complex suggests that there are still professors and buildings that I have omitted from mention only because of lack of time. Occasionally a "look back" on our history and forefathers is a good idea. In writing up my wife's history last week, I found an appropriate quote by one of her ancestors, "The truth is that men who do not honor their forefathers deserve not the respect of their own children and generally they do not receive it."

In summary let's reiterate what I may have learned from our Scholars and Saints:

Bohan — "Examine the patient thoroughly — the patient may be neurotic."
Robinson — "Show concern for your patients — even if someone else has to get up at night."
Murphy — "Pay attention to Kansas farmers — they're the salt of the earth."
Wescoe — "Never write a memo — it may be used against you."
Latimer — "The addition of color to pictures may make it prettier — but not necessarily more accurate."
Tracy — "The word of the professor may or may not be the gospel truth."
Francisco — "If you're in trouble — don't blame it on the professor."
Clendening — "Don't put your heart and soul into your teaching — you may end up in jail."
Hertzler/Hashinger — "When in doubt, take it out — the internist may need another patient."
Calkins — "Put numbers on it — people may believe you."
Peete — "Try, at least, to know the names of your students."
Wahl — "Don't depend on the bureaucracy — do it yourself."
Major — "Try to read a bit each week — even if it's out of print." "If you are unhappy, declare an emergency and act on it." "It's O.K. to dream a bit — but it won't get you anywhere." "News of beer drinking travels fast among students — avoid it."
Orr — "Patients are the ultimate source of information — study them, then operate them." "Surgeons should avoid making speeches."

A beautiful Kansas sunset, "Far above the golden valley," reminds me to thank you again for electing me to be your "Kansas Son."

Reference

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Acknowledgement

Helen Sims assisted in the preparation of this article.

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Before prescribing, please see full prescribing information. A Brief Summary follows:

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in external and internal hemorrhoids, proctitis, papillitis, cryptitis, and fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol™ Suppositories or Ointment.

CONTRAINDICATIONS

Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

WARNINGS

The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

PRECAUTIONS

General

Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

DOSAGE AND ADMINISTRATION

Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

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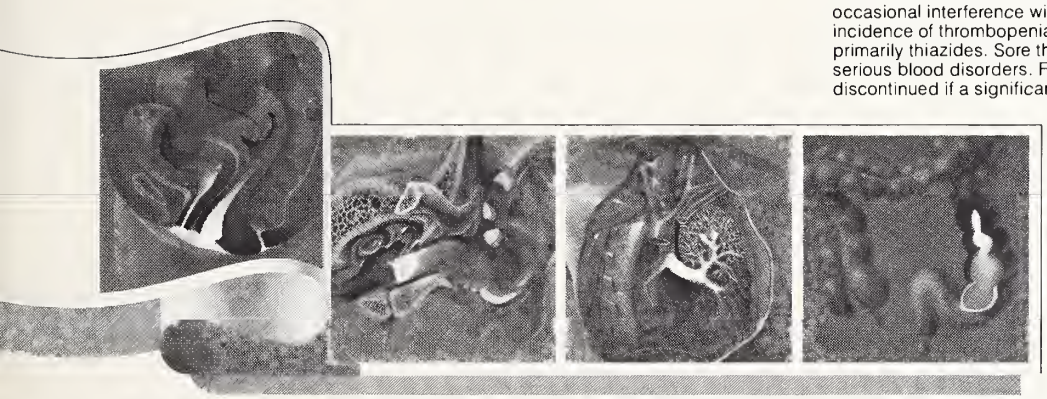
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Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. Limited clinical information presently available on effectiveness of treatment of otitis media with Bactrim when infection is due to ampicillin-resistant *Haemophilus influenzae.* To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

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Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

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1. Rubin RH, Swartz MN: *N Engl J Med* 303:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

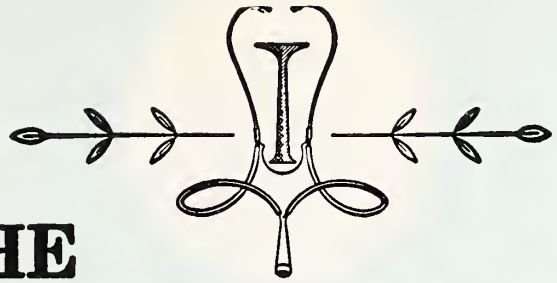
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123rd Annual Session

KANSAS MEDICAL SOCIETY

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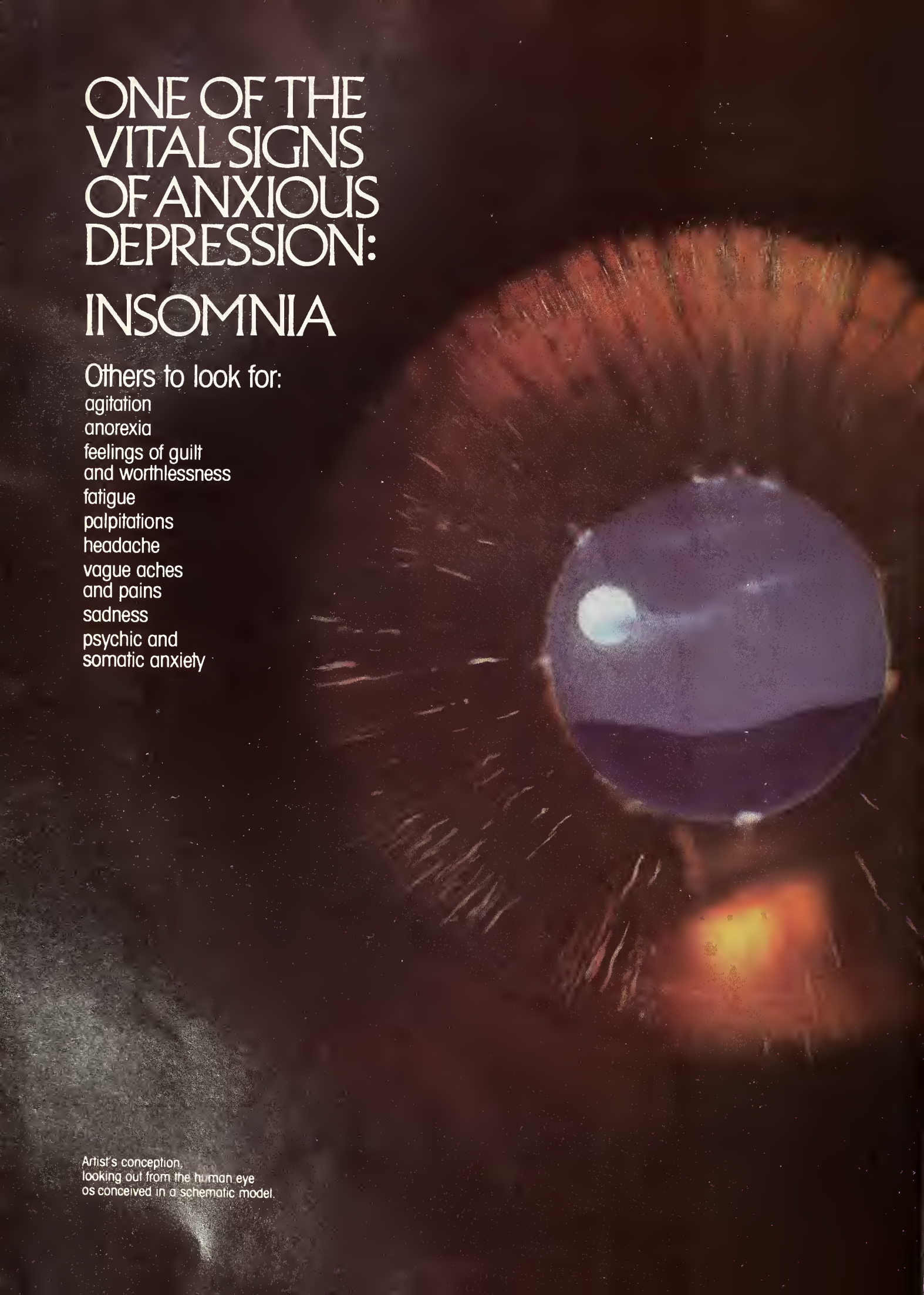
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agitation
anorexia
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fatigue
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headache
vague aches and pains
sadness
psychic and somatic anxiety



Artist's conception, looking out from the human eye as conceived in a schematic model.

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Many patients respond readily to a single bedtime dose of Limbitrol, a convenient schedule that may enhance compliance and helps relieve the insomnia associated with anxious depression. Limbitrol also offers a choice of other regimens: t.i.d., or a divided dose with the larger portion h.s. In all cases, caution patients about the combined effects with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as driving or operating machinery.

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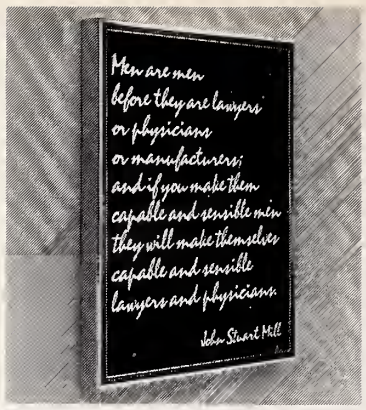
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Please see summary of complete product information on following page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).
***Usage in Pregnancy*: Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude toxic, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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*Reference: 1981/82 *American Druggist Blue Book*

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A Tonic for Geriatric Patients

A pleasant tasting tonic containing iron, vitamins, minerals, and an analeptic. Ideal for those who may benefit from vitamin deficiency prevention. Just one tablespoon before each meal.

DESCRIPTION Forty-five milliliters of SU-TON contains the following ingredients: Pentylenetetrazol, 30 mg • Niacin, 50 mg • Vitamin B-1, 10 mg • Vitamin B-2, 5 mg • Vitamin B-6, 1 mg • Vitamin B-12, 3 mcg • Manganese (as Manganese Sulfate), 1 mg • Magnesium (as Magnesium Sulfate), 2 mg • Zinc (as Zinc Sulfate), 1 mg • Iron (as Ferric Pyrophosphate, Soluble), 22 mg • Alcohol, 18%

INDICATIONS AND USAGE SU-TON contains pentylenetetrazol which may be helpful in the older patient as an analeptic agent when mental confusion and memory defects are present. SU-TON also contains vitamins, trace minerals, and iron, for those patients who may benefit by preventing the development of a deficiency.

CONTRAINDICATIONS Epilepsy, convulsive disorders or known history of sensitivity to any of the listed active ingredients.

WARNINGS The safety of this preparation during pregnancy and lactation has not been established. Use of this drug requires that the physician evaluate the potential benefits of the drug against any possible hazard to the mother and child.

PRECAUTIONS Although there are no absolute contraindications to pentylenetetrazol, it should be used with caution in epileptic patients or those known to have a low convulsive threshold or a focal brain lesion. Caution should be exercised when treating patients with high doses of SU-TON who have heart disease. While pentylenetetrazol does not act directly on the myocardium; the results from central vagal stimulation could cause bradycardia.

ADVERSE REACTIONS Pentylenetetrazol in high doses may produce toxic symptoms typical of central nervous system stimulants, which act on the higher motor centers and the spinal cord. Convulsions resulting from this drug are spontaneous and are not induced by external stimuli. They usually last for several minutes and are followed by profound depression and respiratory paralysis. Death has been reported from the ingestion of 10 grams of pentylenetetrazol.

DRUG ABUSE Drug dependence has not been reported with SU-TON.

OVERDOSAGE Signs and symptoms of acute overdose may be due principally from overstimulation of the central nervous system and from excessive vasodilatation with resulting autonomic nervous system imbalance. The symptoms may include the following: vomiting, agitation, tremors, hyperreflexia, sweating, confusion, hallucinations, headache, hyperpyrexia, tachycardia. Treatment consists of appropriate supportive measures. If signs and symptoms are not too severe and the patient is conscious, gastric evacuation may be accomplished by induction of emesis or gastric lavage. Intensive care must be provided to maintain adequate circulation and respiratory exchange.

DOSAGE AND ADMINISTRATION One tablespoontul (15 ml) 3 times a day 20–30 minutes before meals. This drug is not for use in children under 12 years of age.

HOW SUPPLIED Bottles of 473 ml (16 fl. oz.) NOC 0524-1015-16

Federal law prohibits dispensing without prescription. February 1982

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HAIL, *Steve Carter . . .*



Steven D. Carter assumed the position of Executive Director of KMS on April 5. A native of Holton, Steve holds a B.A. in Communication Arts from Washburn University and a Master of Public Administration from the University of Kansas. He comes to the Society from a position as director of operations for the Kansas Supreme Court.

. . . and FAREWELL, *Jerry Slaughter*

Jerry Slaughter, who has been with KMS since 1973, serving as Executive Director since June 1, 1975, has resigned. He has established his own business, Kansas Management Services, a multi-management firm for small associations. Jerry has contracted with KMS to continue as our governmental liaison.



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They work so well together.



One of man's most amazing explorations and scientific adventures, the successful Gemini flight program was a triumph of imagination and—teamwork. Two men learned to operate in space, to rendezvous, to dock, and to work outside their spacecraft in the hard vacuum of outer space. Not only did they coordinate their efforts with ground backup, they also complemented each other's activities within the close confines of the space capsule.

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☐ *Emollient, for easier bowel movements and soothing relief of local trauma*

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The #1 hemorrhoidal pad† for added external relief and gentle cleansing of fecal residue

☐ *Soothes, cools, comforts the irritation and itch of hemorrhoids and other common anorectal disorders*

☐ *Hygienic rectal wipe—an integral part of the anorectal regimen*

Once pain and inflammation subside, for dual action recommend regular ANUSOL®—to maintain patient comfort—and TUCKS®—to maintain patient anorectal hygiene.

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**Meeting of Am Soc Colon/Rectal Surgeons, May 1980.*
***Based on total prescriptions filled for hemorrhoidal preparations during the first three quarters of 1981. The National Prescription Audit, IMS America Ltd, Sept 1981.*
†1981 data from leading marketing research organization.

PD-85-JA-0867-P-1 (2-82)

ANUSOL-HC® Suppositories/ ANUSOL-HC® Cream

Before prescribing, please see full prescribing information. A Brief Summary follows.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in external and internal hemorrhoids, proctitis, papillitis, cryptitis, and fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

CONTRAINDICATIONS

Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

WARNINGS

The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

PRECAUTIONS

General

Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

DOSAGE AND ADMINISTRATION

Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

Store between 59°-86°F (15°-30°C)

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NOMINATING COMMITTEE

The Nominating Committee of the Kansas Medical Society, consisting of Phillip A. Godwin, M.D., Chairman; Drs. L. William Halling, Warren Meyer and John Yulich, submits to the House of Delegates the following list of nominations for the elective offices of the Society. Wherever more than one nomination appears, they are presented in alphabetical order.

President

Kermit G. Wedel, M.D., Minneapolis. Born in 1932. Graduated from the University of Kansas School of Medicine in 1960. Is in Family Practice. Has served as Councilor. Is currently serving as President Elect.

President Elect

Jimmie A. Gleason, M.D., Topeka. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Obstetrics and Gynecology. Is now First Vice President and has chaired the Legislative Committee for many years.

First Vice President

F. Calvin Bigler, M.D., Garden City. Born in 1931. Graduated from Yale University School of Medicine in 1957. Practices General Surgery. Is Past President of the Kansas chapter, American College of Surgeons, and is now serving as Second Vice President of KMS.

Second Vice President

Clair C. Conard, M.D., Dodge City. Born in 1927. Graduated from the University of Kansas School of Medicine in 1955. Practices Internal Medicine. Now serves as AMA Delegate and Speaker of the KMS House of Delegates.

Jack R. Cooper, M.D., Shawnee Mission. Born in 1917. Graduated from Ohio State University School of Medicine in 1943. Practices Neurosurgery. Is currently serving as Secretary.

Lew W. Purinton, M.D., Wichita. Born in 1923. Graduated from the University of Kansas School of Medicine in 1948. Practices Internal Medicine. Is former chairman of the Commission on education, and is now serving as AMA Alternate Delegate.

Constitutional Secretary

K. William Bruner, Jr., M.D., Wichita. Born in 1944. Graduated from Harvard Medical School in 1970. Practices Pathology. Is active in Society Committee work.

Virginia T. Gruendel, M.D., Kansas City. Born in 1930. Graduated from the University of Kansas School of Medicine in 1955. Is in Family Practice. Is active in Society Committee work.

Wayne O. Wallace, Jr., M.D., Atchison. Born in 1936. Graduated from the University of Missouri School of Medicine, Columbia, in 1965. Is in Family Practice. Is currently serving as Councilor and as Chairman of the Drug Utilization Review Committee.

Treasurer

Richard Meidinger, M.D., Topeka. Born in 1939. Graduated from the University of Kansas School of Medicine in 1965. Practices Diagnostic Roentgenology. Has served as Councilor.

William K. Walker, M.D., Sedan. Born in 1928. Graduated from the University of Kansas School of Medicine in 1945. Is in Family Practice. Is now serving as Treasurer.

Speaker

G. Rex Stone, M.D., Manhattan. Born in 1929. Graduated from the University of Kansas School of Medicine in 1954. Practices General Surgery. Is now Vice Speaker.

Vice Speaker

Franklin G. Bichlmeier, M.D., Kansas City. Born in 1933. Graduated from the University of Kansas School of Medicine in 1958. Practices Cardiovascular Surgery. Is currently serving as Chairman of the Judicial Committee.

AMA Delegate

Donald D. Goering, M.D., Coldwater. Born in 1931. Graduated from the University of Kansas School of Medicine in 1956. Is in Family Practice. Is a Past President of KMS and Kansas Academy of Family Practice, and is currently serving as Chairman of the Member Recruitment Committee.

Alex Scott, M.D., Junction City. Born in 1923. Graduated from the University of Wisconsin Medical School in 1948. Is in Family Practice. Is now serving as AMA Delegate.

AMA Alternate Delegate

Warren E. Meyer, M.D., Wichita. Born in 1927. Graduated from the Northwestern University Medical School in 1951. Practices General Surgery. Is a Past President of KMS.

Linda D. Warren, M.D., Hanover. Born in 1944. Graduated from the University of Kansas School of Medicine in 1970. Is in Family Practice. Is active in KMS and AMA Committee work.

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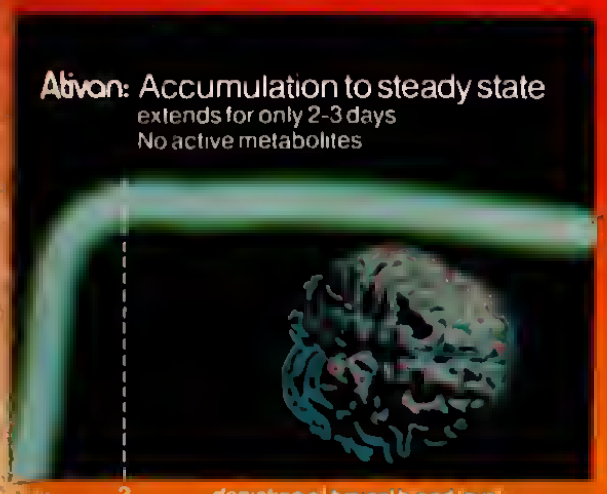
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...there are diagnostic resources and surgical techniques undreamed of only a few years ago, biomedical engineering, new insights also into the action of drugs.

In this changing environment, the way you are practicing medicine is changing too...

Twenty years ago, the benzodiazepines represented a real step forward in the management of anxiety and tension states. In recent years, however, concern about drug accumulation and clearance has led physicians to re-evaluate their use of these agents. In light of current knowledge, many clinicians are changing from multi-metabolite benzodiazepines to Ativan® (lorazepam)—a metabolically and pharmacokinetically distinctive agent that offers clinical advantages which more closely meet the expectations of a modern anxiolytic.

Ativan®: Agent of Change
(lorazepam)







because...

it : shorter acting, v ith less accumulation*

I ntrast to long-acting benzodiazepines, A n has a short, 12-hour half-life, and no a e metabolites In multiple-dose therapy, A n accumulates for only two to three days b re reaching steady state; the long-acting b zodiazepines—diazepam CIV, c diazepoxide CIV, clorazepate CIV and p epam CIV—with their active metabolites— a mulate for as long as 20 days, increasing t elihood of excessive sedation.

it doesn't interact with drugs metabolized by P450 microsomal enzymes

Most benzodiazepines undergo oxidative metabolism and thus utilize the hepatic microsomal enzyme system. Ativan® (lorazepam), however, is metabolized by glucuronidation and does not compete with other drugs for cytochrome P450. Thus, when Ativan is given with Tagamet® (cimetidine), for example, clearance is not delayed, nor sedation increased†—unlike reported observations with patients on other benzodiazepines.[1-5]

it gives you greater control of therapy

The short half-life of Ativan® facilitates more rapid response to dosage adjustments, allowing you to titrate therapy to patients' changing needs. Also, once you decide to discontinue Ativan, it will be out of your patient's system four days after the final dose—in contrast to long-acting benzodiazepines and their active metabolites which take as long as two weeks to be totally eliminated

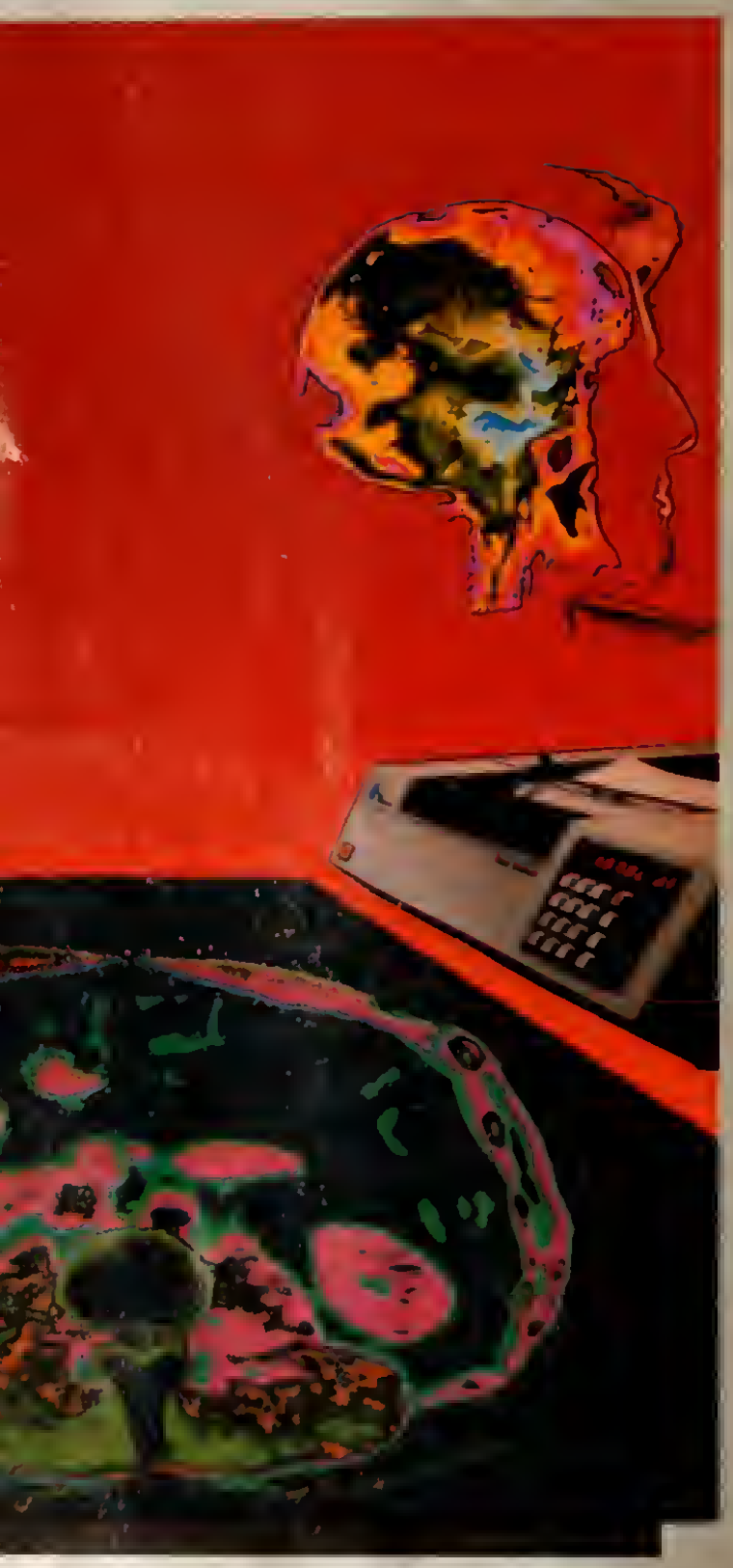
Ativan®
for (lorazepam)IV
Anxiety

See important information on following page

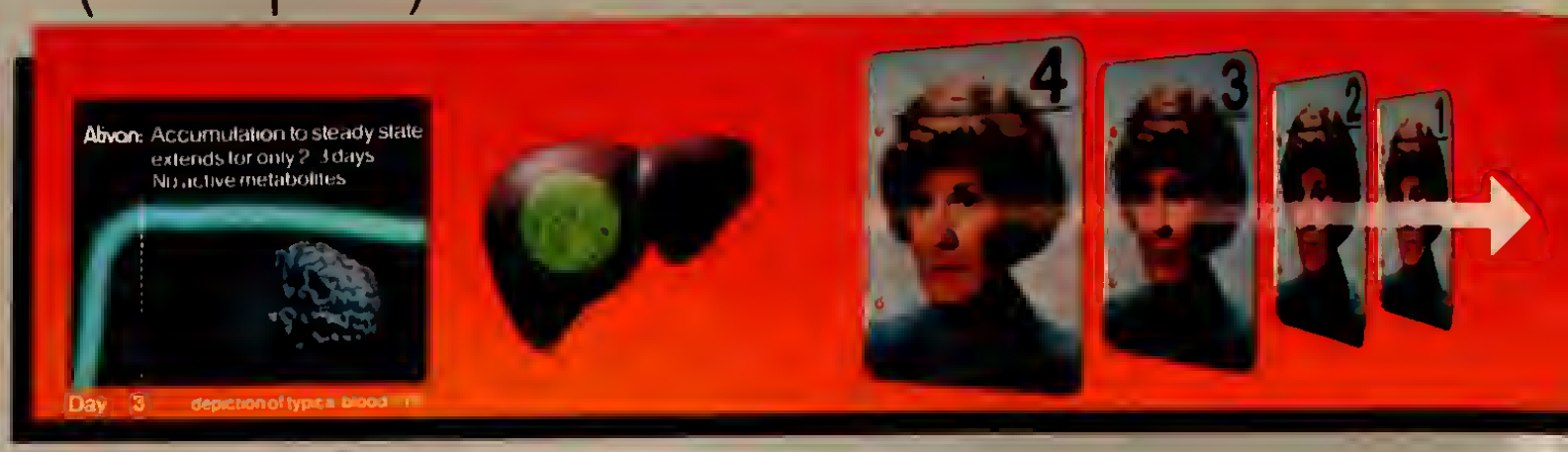
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5 Halo RL, Thompson JF, Segal JL South Med J 74 1075-1078 1981

* armacokinetics cannot as yet be directly related to efficacy

† benzodiazepines produce additive effects when given with CNS depressants such as barbiturates or alcohol



Ativan®: Agent of Change
(lorazepam) ℞



- Little accumulation lessens likelihood of excessive sedation
- Unlike most benzodiazepines, Ativan does not compete with other drugs, such as Tagamet® (cimetidine), for the microsomal enzyme system during biotransformation
- Metabolism not affected by age or liver dysfunction
- Short half-life provides greater control of therapy
- Promptly eliminated from patient's system after discontinuation
- Specifically evaluated and found effective for anxiety associated with cardiovascular and gastrointestinal disorders
- A distinctive change from long-acting benzodiazepines, all of which have active metabolites and are much the same

Brief Summery of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic

Effectiveness in long-term use, i e , more than 4 months, has not been assessed by systematic clinical studies Reassess periodically usefulness of the drug for the individual patient

Contreindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants

Physical and Psychological Dependence. Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e g drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months

Precautions: In depression accompanying anxiety, consider possibility for suicide

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G I disease Safety and effectiveness in children under 12 years have not been established

ESSENTIAL LABORATORY TESTS Some patients have developed leukopenia, some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol

CARCINOGENESIS AND MUTAGENESIS No evidence of carcinogenic potential emerged in rats during an 18-month study No studies regarding mutagenesis have been performed

PREGNANCY. Reproductive studies were performed in mice, rats, and 2 strains of rabbits Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses Clinical significance of these findings is not known However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide

NURSING MOTHERS It is not known if oral lorazepam is excreted in human milk like other benzodiazepines As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15 9%), followed by dizziness (6 9%), weakness (4 2% and unsteadiness (3 4%) Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken Manifestations of overdosage include somnolence, confusion and coma Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U S P Usefulness of dialysis has not been determined

***Dosage*: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

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The Kansas Medical Society Auxiliary

57th Annual Convention, The Hilton Inn East, Wichita

THURSDAY, APRIL 29

9:00 Tennis
Crestview Country Club
10:00 Golf
Wichita Country Club
12:45 Home Computer Seminar
Meet in Lobby
7:00 Cocktails
Crestview Country Club
8:00 Sports Banquet
Crestview Country Club

FRIDAY, APRIL 30

7:45 Registration and Hospitality
Room 104
9:00 Pre-Convention Board Meeting
Oak/Walnut Rooms
10:00 Witness Wichita Tour
Meet in Lobby
12:15 Luncheon Honoring Past Presidents
Center Ballroom
Entertainment by Coleman Junior High Chanticleers
1:30 General Session I
Center Ballroom
4:00 KaMPAC Hospitality Suite
Cottonwood/Pecan Room
Everyone invited
6:30 Cocktails, Banquet, and AMAERF Auction
Holiday Inn Crystal Room

SATURDAY, MAY 1

7:30 Past Presidents' Breakfast
Walnut Room
7:45 Registration and Hospitality
Room 104
9:00 General Session II
Center Ballroom
12:00 Luncheon honoring AMA Auxiliary President Isobel Dvorsky (Harry S.), KMS President Herman W. Hiesterman, M.D., and the KMS Auxiliary Advisory Committee. Home of Dr. and Mrs. S. J. Farha, 16150 East Harry
1:30 Post-Convention Board Meeting
Walnut Room
5:30 Reception for Physicians and Spouses
Sponsored by KU Alumni Association
Wood Rooms
7:00 Annual President's Banquet
Grand Ballroom
Entertainment by the Wichita State University Madrigal Singers. Dance will follow with music by the Dads of Dixieland.

SUNDAY, MAY 2

7:30 Early Bird Breakfast
North Ballroom
Featured Speaker: ***John-Henry Pfifferling, Ph.D.*** "Coping With the Stress of Medical Lifestyle"

IN MEMORY

It is with a deep sense of loss that the Kansas Medical Auxiliary records the names of its members who have died during 1981-1982.

We treasure the memories of these friends and we are grateful for their many contributions to the Auxiliary:

Name	*City*	*County Auxiliary*
Betty Belle Butin (J. Walker)	Wichita	Sedgwick
Laura Campbell (John)	Pratt	Pratt
May Colt (James D., Sr.)	Manhattan	Riley
Sonia George (M. Donald)	Wichita	Sedgwick
Pat Hinshaw (Alfred H.)	Wichita	Sedgwick
Lenore Peters (Glenn R.)	Kansas City	Wyandotte
Phyllis Ripley (Sherman G.)	Salina	Saline

KMS Auxiliary Officers 1981-82

President	Betty Moore (Robert R.)
President-Elect	Meldon Laury (David G.)
First Vice President	Diane Sanders (J. Alan)
Second Vice President	Ann Rempel (John)
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Treasurer	Caryl Bichlmeier (Frank)
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Parliamentarian	Lela May Young (Chester)

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J. Alan Sanders, M.D.	Lawrence
Kermit Wedel, M.D.	Minneapolis
Frank Bichlmeier, M.D.	Shawnee Mission

KMA Convention Committee — 1982

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TELEPHONE NUMBERS

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Hilton Inn	686-7131
Holiday Inn-East	685-1281
Crestview Country Club	733-1344
Wichita Country Club	682-5566

GENERAL INFORMATION

Locations are in the Hilton Inn except where otherwise noted.

Thursday, April 29

8:00 Registration
Lobby
10:00 Impaired Physician Committee
Center Ballroom
9:30 Tennis
Crestview Country Club
1:00 Golf
Crestview Country Club
7:00 Cocktails
Crestview Country Club
8:00 Sports Banquet
Crestview Country Club

Friday, April 30

7:00 Kansas Foundation for Medical Care
Cottonwood/Pecan Room
7:30 Registration
Lobby
9:00 First House of Delegates
South Ballroom
11:30 Kansas OB-GYN Society
Cottonwood/Pecan Room
12:30 Delegates Luncheon
North Ballroom
2:00 Reference Committee
South Ballroom
4:00 KaMPAC Hospitality Suite
Cottonwood/Pecan Room
6:30 Kansas Medical Auxiliary Dinner & Auction
Holiday Inn-East
8:00 American Association of Physicians & Surgeons
Oak/Walnut Room

Saturday, May 1

6:30 "Fun Run"
Lobby
7:30 Registration
Lobby
7:30 Past Presidents Breakfast
Oak Room
8:30 "Health and Care of the Aging"
South Ballroom
9:00 Kansas Chapter, American Academy of Pediatrics
Cottonwood/Pecan Room
12:00 Section on Ear, Nose & Throat
Oak Room
5:30 Reception for Physicians & Spouses
Wood Rooms
7:00 Annual President's Banquet
Grand Ballroom

Sunday, May 2

7:30 Registration
Lobby
7:30 Early Bird Breakfast
North Ballroom
9:00 Second House of Delegates
South Ballroom
12:00 Kansas Allergy Society
Judges Chambers
12:00 Section on Nuclear Medicine
Maple Room
12:00 American Medical Directors Association
Cottonwood/Pecan Room
12:30 Council Luncheon and Meeting
Oak/Walnut Room

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Welcome to Wichita

As President of the Sedgwick County Medical Society, I wish to extend to all members of the Kansas Medical Society and their spouses a cordial welcome to the annual meeting of the Kansas Medical Society and its Auxiliary, April 29-May 2, 1982, at the Wichita Hilton East.

Our members and Auxiliary have arranged a very fine program for the meeting and we all are honored to act as hosts. We hope that you can join us during that time.

W. G. Cauble, M.D., President
Sedgwick County Medical Society

Hosts for the Meeting

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A. Rex Boyd, M.D., Immediate Past President

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Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

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Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

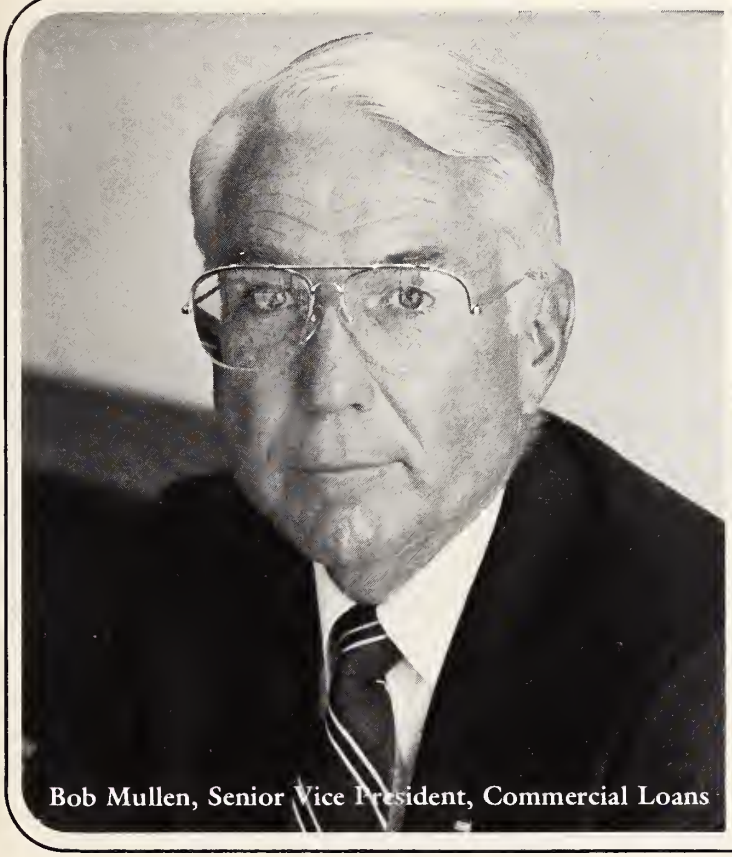
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Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
Robert P. Hudson, M.D., Kansas City ... (913) 588-7040
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
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House of Delegates

Hilton Inn

FRIDAY—APRIL 30

7:30 Registration of Delegates
9:00 First Session

SUNDAY—MAY 2

7:30 Registration of Delegates
9:00 Second Session

Council Meeting and Luncheon at Conclusion of House of Delegates

Reference Committee

Friday, April 30—2:00 p.m.

Educational Grants

The Kansas Medical Society is grateful for the convention program grants received from:

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Distinguished Speakers





WILLIAM G. ECKERT, M.D.

Dr. Eckert, born in New Jersey in 1926, received his medical education at Belleview Medical School, New York University. He gained medical experience in military service, and served a pathology residency in New Orleans. He is currently Deputy Coroner for Sedgwick County and Associate Director of Laboratories, St. Francis Hospital, in Wichita.

He has done extensive research and made numerous presentations in the field of legal and forensic medicine throughout the world for more than 30 years. He has been President of the International Association of Forensic Sciences, Secretary of the American Academy of Forensic Sciences and Secretary-Treasurer of the Kansas Coroners Association, and is immediate past President of the National Association of Medical Examiners. He has served as editor for several publications, and has contributed extensively to various media in the field of forensic science.

Dr. Eckert's topic will be *Medical Legal Aspects of Aging*.

BRUCE N. GARRETT, M.D.

Dr. Garrett was born in Philadelphia in 1947, and received his medical education at The Medical College of Pennsylvania. He subsequently served internship, residency, and fellowships at hospitals in Rhode Island and Texas, prior to his appointment to his present position as Assistant Professor of Medicine at George Washington University School of Medicine, Washington, DC.

His professional activities include membership in several medical societies and contributions to various medical publications.

Dr. Garrett's topic will be *Old and New Concepts in the Treatment of Hypertension in the Elderly*.

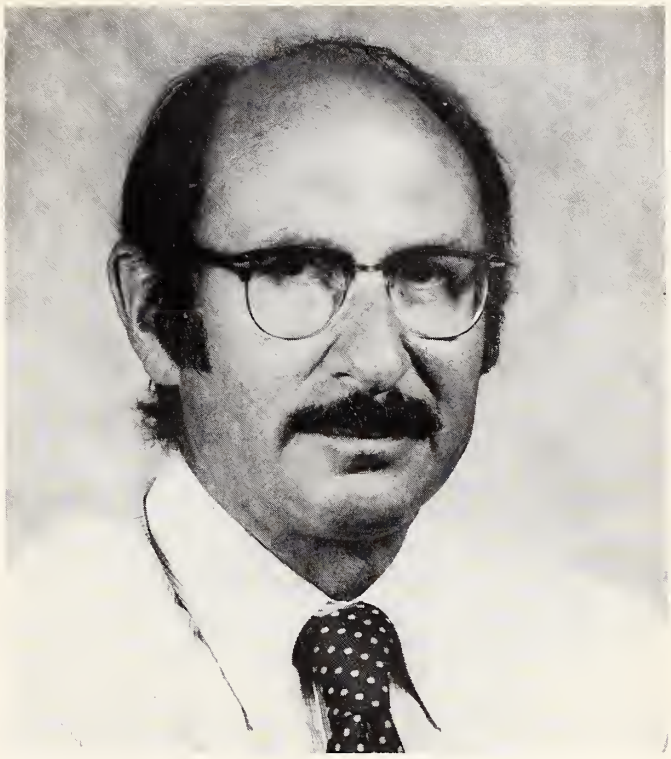


ROBERT T. KELLY, M.D.

Dr. Kelly was born in Minnesota in 1926, and was graduated from the University of Minnesota School of Medicine. He completed his internship at Hennepin County Medical Center.

A charter Diplomate of the American Board of Family Practice, he currently practices with a nine-man multispecialty group in Grand Rapids, Minnesota. He has been active in AMA affairs, and currently is an AMA Commissioner to the Joint Commission on Accreditation of Hospitals. He has been involved in various professional and community activities.

Dr. Kelly will address the first House of Delegates.



ROBERT R. KOHN, M.D.

Dr. Kohn was born in Cleveland in 1925. He earned a Ph.D. in zoology from the University of Michigan and an M.D. from Case Western Reserve University School of Medicine, with additional educational work in biology, pathology, and genetics. At present, he is Professor of Pathology at Case Western Reserve University in Cleveland.

He has been active in research on aging processes, and has made extensive contributions to the literature in this field. He is a member of the Gerontological Research Center, Resources Advisory Committee of the National Institutes of Health, and has served on the Board of Scientific Counselors, National Institute on Aging.

Dr. Kohn will discuss *The Pathology of the Aging Process*.

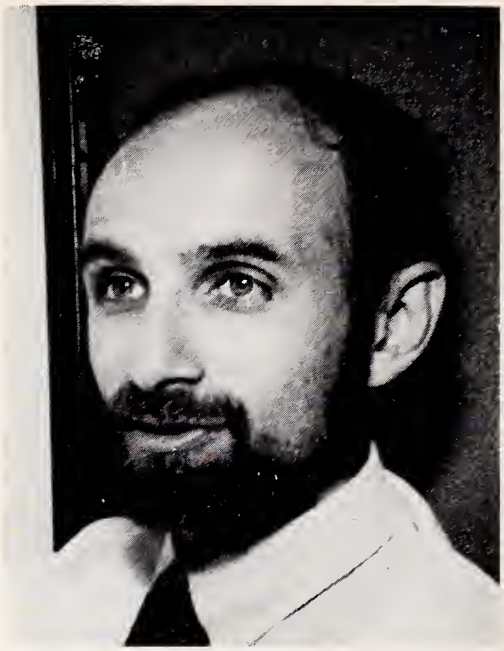


JOHN-HENRY PFIFFERLING, Ph.D.

Dr. Pfifferling is Co-director of the Center for the Well-Being of Health Professionals located in Chapel Hill, North Carolina. He earned his doctorate in medical anthropology and health education from Pennsylvania State University, and completed a post-doctoral fellowship in psychiatry and medicine at Duke Medical Center.

He has lectured and conducted workshops and courses for many organizations of professional health care personnel. He has authored numerous publications, and most recently has addressed the problems of professional impairment. He serves as consultant to a number of organizations concerned with the well-being of health care professionals.

Dr. Pfifferling will address the Early Bird Breakfast. His topic will be *Coping With the Stress of Medical Lifestyle*.



SHELDON H. PRESKORN, M.D.

Dr. Preskorn was born in Wichita in 1948. He was graduated from the University of Kansas School of Medicine in 1974, and subsequently completed residencies in pathology, psychiatry (with internal medicine included) in the Kansas City area prior to serving a residency in psychiatry at Washington University Medical Center in St. Louis. He is currently Assistant Professor of Psychiatry and Pharmacology, UKSM-KC.

He has been the recipient of a number of awards and fellowships, and is active in numerous professional organizations. He has made substantial contributions to medical literature.

Dr. Preskorn will speak on *Geriatric Psychopharmacology, Benzodiazapines: Clinical Relevance of Pharmacokinetics,* and *Movement Disorders in the Elderly*.

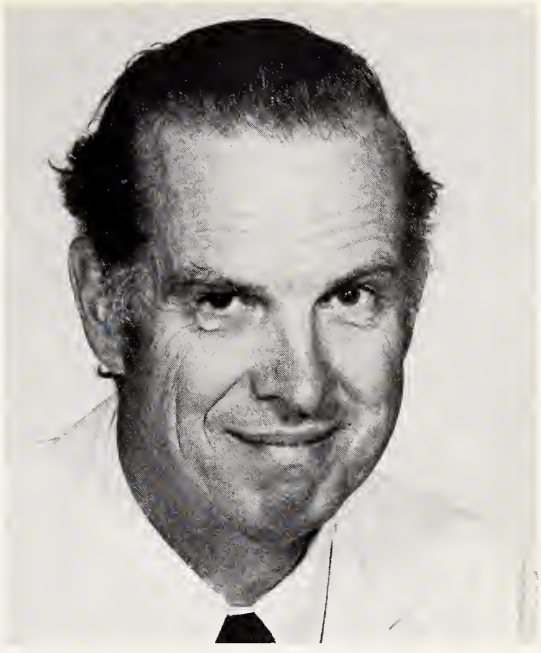


WILLIAM J. REALS, M.D.

Dr. Reals, born in South Dakota in 1920, received his medical education at the Creighton University School of Medicine in Omaha. He presently is Dean of the University of Kansas School of Medicine-Wichita, and recently was elected to a position on the Council on Medical Education of the American Medical Association.

A recognized authority in the field of pathology, he has been extensively involved in organized medicine at the local, state and national levels, and has authored books and numerous articles. He has served in the Army, Air Force and Air Force Reserve, and has been a consultant for a number of federal agencies. He is a past president of the Kansas Medical Society.

Dr. Reals will address the Delegates Luncheon.



IAN MACLEAN SMITH, M.D.

Born in Scotland in 1922, Dr. Smith received his medical education in that country prior to serving various internships and residencies in Glasgow, London, and St. Louis. He is currently Director of the Geriatric Program and Professor of Medicine and Family Practice at the University of Iowa.

He has been active in a number of professional organizations, and has been an invited speaker at numerous medical meetings. He has served as a reviewer and editorial board member for several professional publications. He has received various honors, awards and grants, and has contributed substantially to the medical literature.

Dr. Smith will discuss *Infections in the Elderly*.

PRESIDENT'S BANQUET MAY 1, 1982

Doctor and Mrs. Herman W. Hiesterman extend a personal invitation to members of the Society and their guests to attend the President's Banquet on Saturday evening, May 1, 1982, at 7:00 PM, in the Grand Ballroom.



Harrison C. Boughton

Entertainment will be provided by the Wichita State University Madrigal Singers under the direction of Harrison C. Boughton, coordinator of the voice and choral department at the University. The Singers are a select ensemble whose repertoire draws from Renaissance through contemporary literature. They have toured extensively in the United States and abroad. Participation in the group is open to undergraduate and graduate students, most of whom are music majors.

Their program will include Negro spirituals, vocal jazz, and some selections from Broadway musicals. A medley of Duke Ellington songs will be choreographed and accompanied by piano, bass, and drums.



THE DADS OF DIXIELAND

Following the program, music for dancing will be provided by the Dads of Dixieland under the direction of Corky Edminster. The Dads are a group of Wichita businessmen, most of whom also have backgrounds in professional music.

Thursday, April 29

8:00 Registration — Lobby
10:00 Impaired Physician Committee
Center Ballroom

Sports Activities

9:30 Tennis, Class A & B
Crestview Country Club
1000 N. 127 East
1:00 Golf Crestview Country Club
7:00 Cocktails
Crestview Country Club
8:00 Sports Banquet

Friday, April 30

7:00 Kansas Foundation for Medical Care
Board of Directors
Cottonwood/Pecan Room
7:30 Registration — Lobby
9:00 First House of Delegates
South Ballroom
11:30 Kansas OB-GYN Society
Luncheon and Business Meeting
Cottonwood/Pecan Room
12:30 Delegates Luncheon
North Ballroom
Speaker: ***William J. Reals, M.D.***
Dean, UKSM-Wichita
2:00 Reference Committee
South Ballroom
4:00 KaMPAC Hospitality Suite
Cottonwood/Pecan Room
Cocktails & Hors d'Oeuvres
Everyone invited (Complimentary)
6:30 Kansas Medical Auxiliary
Dinner and AMAERF Auction
Holiday Inn East
7335 E. Kellogg
8:00 American Association of
Physicians and Surgeons
Oak/Walnut Room

Locations are in the Hilton Inn except where otherwise noted.

Saturday, May 1

6:30 "Fun Run" Meet in lobby. Medical families and guests. 1-10 miles; no clock, no divisions, no places, no awards.
7:30 Registration — Lobby
7:30 Past Presidents Breakfast
Oak Room

SCIENTIFIC PROGRAM

"Health and Care of the Aging"

The proportion of the American population over age 65 has been increasing steadily and will continue to do so. This age group also has a greater need for health care services than other age groups. For these reasons, this year's scientific program will focus on the health and care of the aging.

After attending this program, the participant should be able to:

1. Describe the normal physiology of the aging process;
2. Assess and treat common health problems of the elderly;
3. Discuss the pharmacokinetics of various drugs used in the treatment of the elderly; and
4. Identify legal issues in the medical care of elderly.

Continuing Education Credit

As an organization accredited for continuing medical education, the University of Kansas School of Medicine—Wichita designates this continuing medical education activity as meeting the criteria for six (6) credit hours in Category I of the Physician's Recognition Award of the American Medical Association.

This program is acceptable for six (6) Prescribed hours by the American Academy of Family Practice.

8:30 Welcome and Announcements
8:45 The Pathology of the Aging Process
Robert R. Kohn, M.D.
9:45 Geriatric Psychopharmacology
Sheldon H. Preskorn, M.D.
10:30 Break
10:45 Benzodiazapines: Clinical Relevance of Pharmacokinetics
Sheldon H. Preskorn, M.D.
11:30 Questions and Answers
Drs. Kohn and Preskorn
12:00 Lunch
2:00 Old and New Concepts in the Treatment of Hypertension in the Elderly
Bruce Garrett, M.D.
2:45 Infections in the Elderly
Ian Smith, M.D.
3:15 Break
3:30 Movement Disorders in the Elderly
Sheldon H. Preskorn, M.D.
4:00 Medical Legal Aspects of Aging
William G. Eckert, M.D.
4:30 Questions and Answers
Drs. Garrett, Smith, Preskorn, and Eckert
5:00 Adjourn

This program is supported in part by Sandoz Pharmaceuticals and Dorsey Laboratories.

9:00 Kansas Chapter, American Academy of Pediatrics
Cottonwood/Pecan Room
Clinical Grand Rounds
Open to all interested physicians
12:00 Section on Ear, Nose, and Throat
Luncheon and Business meeting
Oak Room
5:30 Reception for Physicians and Spouses
Sponsored by KU Alumni Association
Wood Rooms
7:00 Annual President's Banquet — Grand Ballroom
Entertainment: ***Wichita State University Madrigal Singers***
Music for dancing by ***Dads of Dixieland***

Locations are in the Hilton Inn except where otherwise noted.

Sunday, May 2

7:30 Registration — Lobby

7:30 Early Bird Breakfast — North Ballroom
Featured Speaker: ***John-Henry Pfifferling, Ph.D.,*** Center for the Well-Being of Health Professionals, Coping With the Stress of Medical Lifestyle

9:00 Second House of Delegates — South Ballroom

12:00 Kansas Allergy Society — Judges Chambers
Luncheon and Business Meeting

12:00 American Medical Directors Association
Cottonwood/Pecan Room
Luncheon and Business Meeting

12:30 Council Luncheon and Meeting — Oak/Walnut Room

TELEPHONE NUMBERS

Wichita Area Code — 316

Hilton Inn	686-7131
Holiday Inn-East	685-1281
Crestview Country Club	733-1344
Wichita Country Club	682-5566

Locations are in the Hilton Inn except where otherwise noted.



Committee REPORTS

AMA Ad Hoc Committee on Women in Organized Medicine

The National Ad Hoc Committee on Women in Organized Medicine was appointed by the AMA Board of Trustees and began its work in June 1981. The purpose of the committee is to monitor and to determine ways to increase involvement of women physicians in organized medicine at all levels. Members of the committee are Palma Formica, M.D., New Jersey, Chairman; Marie Kuffner, M.D., Texas; John Madara, M.D., New Jersey; Amilu Martin, M.D., Colorado; Susan Rudd, M.D., Texas; and Linda Warren, M.D., Kansas.

The committee surveyed state, county, and national medical specialty societies to determine the number and percentage of female members and to identify female physicians holding leadership positions. The findings show that nationwide the total number of female members is low (26% of female physicians belong to the AMA as compared to 48% of male physicians). The committee feels that if organized medicine is to properly represent all physicians, the number of female members must be increased to reflect the growing female physician population. The committee sees the strategic link to be *getting the female physician involved at the grass roots level*. Because of this need for local involvement, the committee has worked closely with the AMA Membership Department as well as making specific recommendations to local societies for recruiting female members. Specific target areas for membership have been identified.

The female AMA membership in Kansas (39.6%) is above the national average. However, the Kansas male physicians also are above the national average, with a membership rate of 61.3 per cent. Kansas also is a leader in attempting to involve female physicians, being one of only six states with a committee to address female membership and involvement in organized medicine (chaired by Joan Sehdev, M.D.).

The national committee has found that many women who wish to be active in organized medicine lack basic leadership skills. The committee has recommended to the Board of Trustees that training in these basic skills be made available through the Federation to all physicians — male, female, young, foreign-trained — any physician who feels a lack of skills necessary to an optimal contribution to organized medicine. Some states are beginning to promote this type of leadership training project.

When the committee members met with the Board of Trustees at the 1981 interim AMA meeting, they emphasized two goals to be achieved if organized medicine is to adequately represent the female physician:

- Sincere commitment to the inclusion of women in the mainstream of organized medicine primarily by promoting them to and for leadership positions; and
- Serious attention and effort, based on targeting and marketing, in recruiting women members.

It is the feeling of the committee members that the AMA Board of Trustees has been most receptive to its recommendations, and that the Federation is sincerely concerned that women be mainstreamed into active involvement in organized medicine. Now it is the job of current members, both male and female, to actively recruit non-member female physicians so that the AMA will continue to truly be the voice of the American physician.

—Linda D. Warren, M.D., *Member*
AMA Ad Hoc Committee on Women
in Organized Medicine

Specialty Societies

Friday, April 30

11:30 Kansas OB-GYN Society
Cottonwood/Pecan Room
Luncheon and Business Meeting

8:00 American Association of Physicians and Surgeons
Oak/Walnut Room

Saturday, May 1

9:00 Kansas Chapter, American Academy of Pediatrics
Cottonwood/Pecan Room
Clinical Grand Rounds
All interested physicians welcome.

12:00 Section on Ear, Nose, and Throat
Oak Room
Luncheon and Business Meeting

Sunday, May 2

12:00 Kansas Allergy Society
Judges Chambers
Luncheon and Business Meeting

12:00 Section on Nuclear Medicine
Maple Room
Business Meeting

12:00 American Medical Directors Association
Cottonwood/Pecan Room
Luncheon and Business Meeting

TELEPHONE NUMBERS

Wichita Area Code — 316

Hilton Inn	686-7131
Holiday Inn-East	685-1281
Crestview Country Club	733-1344
Wichita Country Club	682-5566

Councilor Reports

Activities in the Council Districts of Kansas

DISTRICT 1

The meeting of the First Council District of the Kansas Medical Society was held at the Atchison Hospital, Atchison, on Sunday, November 3, 1981.

Jerry Slaughter, Executive Director of the KMS, gave a presentation informing the membership of the successes and failures of the Medical Society in achieving its goals with the Kansas Legislature. An update on the status of the lawsuit filed by the KMS *vs* The Kansas State Board of Nursing was also presented.

Emerson Yoder, M.D., Denton, former President of KMS, was in attendance.

Wayne O. Wallace, Jr., M.D., Atchison, was elected Councilor for District 1.

WAYNE O. WALLACE, JR., M.D. *Councilor*

DISTRICT 2

For the first time in many years, Wyandotte County Medical Society gained more members than it lost, going from 385 voting members in December 1980 to 401 voting members in December 1981. The efforts of KMS President Herman W. Hiesterman, M.D., to encourage medical students and residents to join organized medicine are producing results in our area. Since January 1, we have received applications for membership from 29 medical students and residents.

Our president-elect, Jack D. Walker, M.D., was forced to resign during 1981 for reasons of health. We were fortunate to obtain David M. Pugh, M.D., as his replacement, and Dr. Pugh is doing an exemplary job as President this year.

Wyandotte County Medical Society presented a plaque to Henry L. Regier, M.D., in celebration of his 100th birthday on September 30, 1981. Dr. Regier practiced medicine in Kansas City from 1910 until 1971, when he retired; he served as President of WCMS in 1935.

Our Emergency Medical Care Committee continues to be very active, working with the mayor to continually upgrade the EMS system. As a result of these efforts, the city has embarked upon a program of training firefighters as EMTs. Eventually, all Kansas City, Kansas, firefighters will be trained as EMTs.

The Children and Youth Clinic has been operated for 13 years by the Department of Pediatrics at UKSM-KC with funding from a federal grant. This grant was substantially reduced for 1982. The Department of Pediatrics felt it could not continue the clinic with these budgetary restraints, and the City-County Health Department was offered the reduced grant. The Public Health and Welfare Committee and the Board of Directors of the Medical Society opposed this move on the basis that this would mean that the Health Department would be providing primary ambulatory care which would be a deviation from their stated purpose of preventive medicine. After several months of discussions, the Board of Health voted to sign the contract to provide this service despite the Medical Society objections.

The Wyandotte County Medical Society greatly appreciated the efforts of Dr. and Mrs. Herman W. Hiesterman in braving the bad weather to come to Kansas City on December 15, 1981, to address the Medical Society.

LOUIS M. CULP, M.D., *Councilor*

DISTRICT 3

The Johnson County Medical Society continues its rapid growth, adding 21 active and eight corresponding members during 1981. This brings the total membership as of January 1, 1982, to 374, and represents a net gain of 22 members for 1981. Active membership in the society has increased 80 per cent since January 1975.

The monthly meetings continue to be well attended, with programs planned to be of interest to all physicians. During the past year, program topics have included presentations and discussions of the Kansas Foundation for Medical Care, Blue Cross/Blue Shield, Individual Practice Associations, problems faced by the University of Kansas Medical Center, Health Care Stabilization Fund, and professional liability problems.

A liaison committee with UKSM-KC has been established to develop an improved understanding of those problems and concerns of mutual interest involving the Medical Center and the medical community.

A substantial contribution was made to the Ronald McDonald House, a nonprofit housing facility for the families of children with malignancies undergoing therapy at the Medical Center.

The society is currently sponsoring a KMS communications project consisting of taped interviews with physicians on various medical topics to be used as a means of furthering public education concerning health care. Members of the Medical Society Auxiliary will handle the interviews.

James G. Bridgens, M.D., *Councilor*

DISTRICT 4

Council District 4 entertained the Kansas Medical Society President, Herman W. Hiesterman, M.D., on February 9, 1982. The meeting included an interesting discussion. A good representation of the membership was present.

George W. Pogson, M.D., *Councilor*

DISTRICT 5

In September, Herman W. Hiesterman, M.D., President of the Kansas Medical Society, was the guest speaker at the annual Fifth Council District Meeting held in Manhattan. He was accompanied by Mrs. Hiesterman and by Jerry Slaughter, Executive Director of the KMS.

Kenneth M. Boese, M.D., *Councilor*

DISTRICT 6

The Shawnee County Medical Society gained 12 members during 1981; current total membership is 335 physicians. Our 1981 President, Stuart C. Averill, M.D., and our 1982 President, Larry G. Rotert, M.D., have provided effective leadership. One focus has been the attempt to improve lines of communication between the medical community and the business community. A joint meeting between business leaders and physicians was held, and was quite successful. Subsequently, Topeka physicians contributed substantially to the efforts of the local Chamber of Commerce to attract new business growth to the Topeka area.

In March 1981, the Society sponsored a gala meeting in honor of Dr. Lucien Pyle's 50th anniversary in the practice of medicine. Well known to physicians throughout the state, Luke Pyle has been a founding Fellow of the American College of OB-GYN, a diplomate of the American Board of OB-GYN, President of the Shawnee County Medical Society, a long time member of the Editorial Board of *The Journal of the Kansas Medical Society,* and President of the Kansas Medical Society in 1953. During the years, he has contributed a vast amount of his time and expertise to important state government advisory committees and has given of his time extensively as a teacher of nurses and in numerous other educational functions.

The Shawnee County Medical Society recently moved into new headquarters in the Topeka Blood Bank building. A large two-room suite provides room for the activities of our very successful physician referral service and for the compiling and editing of *The Bulletin* of the Society. *The Bulletin* was redesigned during the past year, and we initiated the sale of advertising to local business firms. This policy has permitted *The Bulletin* to function in a largely self-supporting fashion. It is being published every other month.

Other activities, too numerous to mention in detail, included an annual phyician-attorney banquet hosted by the Topeka Bar Association, and the continued successful growth and functioning of the education wing of our Society, the Shawnee County Medical Foundation. The board of directors also provided a position paper to our district judges on the need for a forensic pathologist for the Topeka area.

Finally, due to the efficient work of our board of directors, officers, and most particularly our Executive Director, the Society was able to decrease its dues both in 1980 and 1981.

James H. Ransom, M.D., *Councilor*

DISTRICT 7

District 7 — primarily the Flint Hills District Medical Society — continues to grow and improve in expertise as well as numbers. There are currently 53 members. Those new since last year include an internist, a surgeon, a family physician, a woman pediatrician, a part time dermatologist, and a part time otorhinolaryngologist. New physicians planning to begin practice here this summer include internist James Barnett, M.D., Surgeon Ron Farris, M.D., and psychiatrist Marucio Masferrer, M.D., who will return from Canada.

New Alternate Councilor, John P. Brockhouse, M.D., hosted the President's Dinner in October with KMS President, Herman W. Hiesterman, M.D., and KMS staff. Flint Hills officers this year are Richard L. Frazier, M.D., President; David J. Edwards, M.D., Vice President; Frederick S. Neuer, M.D.,

Secretary-Treasurer; and John F. Best, M.D., Program Chairman.

Programs for the 1981-82 season include the following:

- Dennis Artzer, M.D., Nephrologist, discussed the diagnosis and treatment of essential hypertension, March 1982.
- Mrs. Karen Johnson presented a program on cardiopulmonary resuscitation, February 1982.
- Joseph Bosiljevac, M.D. gave a presentation on carotid artery surgery, January 1982.
- James Bergen, M.D., Chief of Internal Medicine of Bethany Hospital and a clinical professor of medicine at the K. U. Medical Center, gave an interesting slide presentation of unusual cases. Flint Hills Medical Society members were invited to participate in the differential diagnoses of these unusual cases.
- In October 1981, Douglas Amend, M.D., discussed dysmenorrhea.
- The September 1981 meeting was presented by Sean Lynch, M.D., UKSM-KC. He discussed iron deficiency anemia and iron metabolism.

Carl and Theresa Didde of Didde Graphics, Inc., have generously offered the gift of a CAT scanner for installation at St. Mary's Hospital in Emporia. Three capable young radiologists could make this work very well for this area; HSA approval is pending.

MARVIN D. SNOWBARGER, M.D., *Councilor*

DISTRICT 8

KMS President, Herman W. Hiesterman, M.D., attended one of the fall meetings of the Cowley County Medical Society, and Butler-Greenwood County members were invited guests. Although attendance from the latter counties was low, our President gave an excellent discussion of the services, program, and effectiveness of the American Medical Association and encouraged all members present to participate as members of the AMA.

Our location on the Oklahoma border has some advantages relating to the exchange of information. The Oklahoma Medical Society has established its own malpractice insurance for basic coverage, and the plan has been successful. They have now expanded this to cover health insurance for all society members and their employees and families. This has resulted in a substantial savings for members, and it would appear that KMS should explore a joint operation, since the OMS has all of the computers and personnel, and could probably add KMS participants with little expense. I intend to discuss this at the next Council meeting.

As always, there is a reticence on the part of most KMS members to assist with coroner duties. It would be wonderful if each member would resolve to volunteer at least some assistance during the coming year in carrying out the coroner duties. One of our Past Presidents — Dr. John Lattimore, now deceased — was the champion of our present system, but performing the function has fallen on too few of our members.

I hope the entire membership from the Eighth Council District will attend a portion of the Annual Meeting April 29-May 2 in Wichita. See you there!

NEWTON C. SMITH, M.D., *Councilor*

DISTRICT 9

The medical climate in Council District 9 has been stable during the past year. Several new physicians have moved into the area and several other physicians have left. In November 1981, the President of the Kansas Medical Society, Herman W. Hiesterman, M.D., met with the physicians of the District in Salina.

A linear accelerator and a CAT scanner have been added to our district.

The Smokey Hill Family Practice Department has five residents in Salina and will graduate their first class in June 1982.

HERBERT D. DOUBEK, M.D., *Councilor*

DISTRICT 10

District 10 members spent an enjoyable evening at the Prairie Dunes Country Club in Hutchinson with KMS President Herman W. Hiesterman, M.D., and Gary Caruthers on November 24, 1981. An animated discussion followed the presentation by Dr. Hiesterman. Special music was presented by the Deknatel Trio of Bethel College, North Newton, Kansas.

HERBERT FRANSEN, M.D., *Councilor*

DISTRICT 11

January 1981: Rex Boyd, M.D., assumed presidency of the Medical Society of Sedgwick County from Lew Purinton, M.D., outgoing president. Phillip A. Godwin, M.D., President of the Kansas Medical Society, and Jerry Slaughter discussed cur-

rent activities of the KMS and the outlook for legislation by the Kansas Legislature in 1981.

February 1981: At the general membership meeting, representatives of the Wichita Chamber of Commerce discussed the community outlook for growth, economy, and problems in the 1980s. MSSC Legislative Committee, under the chairmanship of K. William Bruner, M.D., renewed committee activities in close liaison with the KMS Legislative Committee concerning health related legislation. The MSSC Legislative Committee held their annual dinner for the legislators from Sedgwick County.

March 1981: Wichita Police Chief Richard LaMunyon discussed drug and crime problems in the city, the need for changes in the current parole system, and an overview of police/community relations. The MSSC Auxiliary presented a gift of $3,683.70 to the MSSC Scholarship Loan Fund, and made an additional contribution of $1,371 to AMAERF. These funds were the proceeds from the annual Christmas card project.

April 1981: Col. John Anderson, Hospital Commander at McConnell Air Force Base, hosted the general membership meeting. Col. Sloan from Vandenburg Air Force Base presented a program on the U. S. Space Shuttle Program. The MSSC membership was saddened by the deaths, during March 1981, of the following physicians: Harry O. Anderson, M.D.; Milton Lindley, M.D.; Clifford E. Jones, M.D. and Larry E. Woodward, M.D.

May 1981: The membership held their annual business meeting. Terry Pling, M.D., was elected president-elect for 1982. The by-laws of the society were amended with identical requirements for membership in the society by MDs and DOs, and to allow physicians outside of Sedgwick County to be members of MSSC. A progress report of MSSC Foundation activities was given. Dr. Bruner reported on legislation enacted by the 1981 Legislature, and the Councilor reported actions of the recent KMS House of Delegates.

No membership meetings were held in June, July, or August 1981. During this time, the Board of Directors approved an amended update of the medical-legal code which is used by the local Joint Medical Legal Committee. Dr. Robert Harder (SRS) discontinued the nursing home utilization review program by MSSC physicians. He also met with members representing hospital administrators, Wichita hospitals and the MSSC Foundation, and requested consideration of a proposal to carry out in-hospital utilization review by the Foundation for Medicaid recipients.

During the summer, the medical community was saddened by the deaths of Louis S. Roberts, M.D.; Harold Brooks, M.D. and Paul Wedin, M.D.

September 1981: The Annual MSSC Sports Day was held with contests in golf, tennis, and racquetball. The Fourth Annual Physicians Awards were made at the Sports Award Dinner. Recipients were: Norton L. Francis, M.D.; Henry Tihen, M.D.; and Donald Trees, M.D.

November 1981: The third annual joint meeting of providers and representatives of labor unions, industry management, and insurance companies was held at the MSSC building. Ivan E. Rhodes, M.D., President of the MSSC Foundation for Medical Care, reviewed the foundation's program for in-hospital utilization under contract for employees and dependents of The Boeing Company. At that time, the program had been in effect for one year, and data from the first six months showed that there was 30 per cent less in-hospital utilization than with similar Blue Cross-Blue Shield and commercial insurance covered patients. The admission rate of 117/1000 subscribers compared with an admission rate of 171 admissions/1000; and with the bed/day utilization of 681 bed days/1000, compared to 1013/1000 for Blue Cross-Blue Shield and commercial subscribers. The contract with The Boeing Company was extended for another year. This project has been very successful, and has been carried out without difficulty thanks to the splendid cooperation of the community hospitals, physicians, health insurance companies under contract, The Boeing Company, and their employees and dependents.

Richard J. Cummings, M.D., was appointed by the Governor to the Board of Healing Arts to complete the unexpired term of William C. Swisher, M.D., who resigned for health reasons.

December 1981: UKSM-Wichita was the subject for the general membership meeting. William J. Reals, M.D., Dean; Lorene Valentine, Frederic C. Chang, M.D. and William R. Levine, M.D., Associate Deans, discussed the history, current status of the school, manpower — Kansas perspective, and a review of admission and student performance at the Wichita campus.

MSSC adopted the AMA policy regarding the use of drugs with weight reduction programs for patients.

January 1982: Wilbur Cauble, M.D., assumed presidency of MSSC from Dr. Rex Boyd. Herman W. Hiesterman, M.D., KMS President, and Jerry Slaughter were guests of the society. Dr. Hiesterman presented a plaque from the KMS to Senator Wesley Sowers of Wichita, as directed by the House of

Delegates in 1981. Mr. Slaughter provided an overview of the possible legislative matters in the health care field for 1982.

February 1982: Robert T. Stephan, Attorney General for the State of Kansas, was the guest speaker at a joint meeting with MSSC members and spouses.

March 1982: The MSSC Foundation for Medical Care will expand its utilization review activities to include three other major employer groups in the Wichita community.

The EMS Advisory Committee, chaired by Robert Tinker, M.D., has been very active during the year in supporting and maintaining guidelines in cooperation with the city/county government to provide quality and efficient EMS service to the community.

The MSSC paging system continues to be very active with more than 450 pagers in operation.

The MSSC Patient Referral System continues to be very active, filling a need in the community by obtaining appointments for individuals who have not established doctor-patient relationships.

The Building and Grounds and Judicial and Finance Committees are working with local architects on plans for expansion of the MSSC office. Target date for beginning construction is 1982.

The MSSC Mixed Bowling League folded quietly after 20 years as physicians and spouses took up more strenuous athletic endeavors.

Some 8,600 people participated in Health Fair '81 at 23 sites. This was a community health screening program in which MSSC participated with the rest of the community.

IVAN E. RHODES, M.D., *Councilor*

DISTRICT 13

Central Kansas Medical Society now has 70 members. Officers for 1982 are: Earl D. Merkel, M.D., Russell, President; Dallas Richards, M.D., Hays, Vice President; and Robert Enberg, M.D., Hays, Secretary-Treasurer. Joining our ranks this year were Shobhana Bhargava, M.D., LaCrosse, and William Dickerson, M.D., and Joel Jacobson, M.D., both of Wakeeney.

KMS President Herman W. Hiesterman, M.D., delivered the annual presidential address in his inimitable and dynamic style at the November meeting of our Society.

Our "tempest in a teapot" this year is the dispute of the local Right to Life *vs* Planned Parenthood groups. We are unique in that we are the only community in Kansas other than Wichita with a Planned Parenthood facility. An improvement over past years is that there is now a local physician advisory committee purportedly to advise the Planned Parenthood personnel on a local level. Still to be addressed are points regarding independent practice of a de facto free-standing paraprofessional and the dispensing of drugs by non-physician, non-pharmacist personnel in this setting.

WALLACE N. WEBER, M.D., *Councilor*

DISTRICT 14

Council District 14 met on November 18, 1981, at the Black Angus Restaurant in Great Bend. Herman W. Hiesterman, M.D., President of the Kansas Medical Society, was guest of honor and delivered the address to the physicians present. Jerry Slaughter, KMS Executive Director, presented a brief update on KMS actions. An appeal was made for physicians to contact their local legislators about the impending 1982 legislative calendar. Members will meet separately with their legislators. A follow-up meeting will be held with the Barton County Medical Society in the spring of 1982.

The meeting concluded with a discussion regarding areas of concern that should be addressed by the Kansas Medical Society. The meeting was adjourned, and Council District 14 will meet again in the fall of 1983.

Most members of the Barton County Medical Society are also staff at the Central Kansas Medical Center. As yet, we have undertaken no major project as a council district.

DONALD E. BEAHM, M.D., *Councilor*

DISTRICT 16

Northwest Kansas Medical Society was honored by the visit of KMS President Herman W. Hiesterman, M.D. (a member of our NWKMS), on September 2. We wish to thank him for bringing us up to date on activities and services provided by the American Medical Association. One hundred per cent AMA membership for NWKMS would certainly seem to be a worthwhile goal as all derive benefits from our national organization.

Membership in NWKMS has remained unchanged during the year. We welcome the following new physicians to our area and hope they will become members of our society: Sudheer V. Mitra, M.D., Goodland; Kyeong Hwan Seo, M.D.,

Norton; and Michael D. David, D.O., Oakley.

JOHN R. NEUENSCHWANDER, M.D., *Councilor*

DISTRICT 17

The Southwest Kansas Medical Society has been increasingly active this past year under the leadership of Frank D. Eichhorn, M.D., President, and Luther L. Fry, M.D., Secretary-Treasurer. Four meetings were held during 1981, all at the new Garden City Country Club, including one scientific session and three meetings with guest speakers. All were extremely well attended.

We had the unusual opportunity of having two presidents of the Kansas Medical Society visit and speak to us during the past year. Phillip A. Godwin, M.D., spoke at our February meeting, and Herman W. Hiesterman, M.D., spoke at our November meeting, so that most of us in Southwest Kansas are truly up to date on national and local medicine and politics. Roger D. Warren, M.D., also spoke at our November meeting and increased our knowledge of and enthusiasm for KaMPAC.

We continue to be blessed with the opportunity to exchange knowledge with medical students and residents from the universities of Kansas and Colorado. At times it is difficult to tell who is the trainee. We hope to attract some of the sharp young medical people to permanently practice in our area so that we can keep up with the rapid population growth. Hopefully hospital expansion and growth will also occur.

Several of our members continue to work closely with the Impaired Physician Program, and we appreciate their anonymous support to colleagues in our area.

MAX E. TEARE, M.D., *Councilor*

DISTRICT 18

The district has been quiet this past year, free from any large problems. In Ottawa, the physicians joined together and conducted a series of radio spots on health topics which were well received by the community. They are in the process of developing a cardiac rehabilitation program at the hospital, and this should be a valuable asset to them.

Members of the Douglas County Medical Society have been appearing on a local radio show for discussions of medical problems, and this has been most informative. The Douglas County Auxiliary conducted a successful fund drive for the Ronald McDonald House. The Auxiliary and the Medical Society both joined with other health agencies in conducting a county health fair during the past year.

No new physicians have moved into the district, but some are anticipated during the coming year.

ROBERT W. HUGHES, M.D., *Councilor*

DISTRICT 19

The monthly meetings of District 19 continue to be successful and well attended. They serve the physicians of this seven-county area of Southeast Kansas in a manner I would describe as educational, social, and political — in that order. (There is little political unrest in the Balkans.)

William W. Emmot, M.D., has done a fine job this past year of arranging the scientific portion of each meeting. Guest speakers from UKSM-KC have been well received.

KMS President Herman W. Hiesterman, M.D., was the featured speaker at the March 17 meeting in Independence, bringing us up to date on the activities of the Kansas Medical Society.

It has been my pleasure to serve this fledgling district as Councilor these past few years. It is now time to turn it over to younger and more energetic hands. The older I get, the more I must remember not to let the practice of medicine interfere with golf and fishing.

ROBERT F. MOORE, M.D., *Councilor*

The Drug Information Service is maintained by the Department of Pharmacy at the K.U. Medical Center to promote rational drug therapy. The Service is staffed by clinically oriented pharmacists with access to accurate, current, and unbiased information. Information is available free of charge to any health care practitioner involved with patient care. Contact:

Drug Information Service
3930 Cambridge
Kansas City, KS 66103

Telephone (any time): 913-588-2328

Kansas Medical Assistants

42nd Annual Convention, Ramada Inn Downtown, Topeka

Down Memory Lane

FRIDAY, APRIL 30

2:00 Registration, Lower Lobby
2:00 Silent Auction, Lower Lobby
3:00 Executive Board Meeting, Parlor B
5:00 Leadership Seminars
8:00 Beer Barrel Polka Party, Lower Lounge
Courtesy Munns Medical Supply

SATURDAY, MAY 1

7:30 KMAS Past Presidents' Breakfast, Green Room
7:30 Rolls and Coffee, Lower Lobby
8:00 Registration/Silent Auction, Lower Lobby
9:00 General Session, Grand Ballroom
Presiding — *Anna Marie Dercher, RT*
Welcome — *Joan Sehdev, M.D.*, Representing KMS
10:30 Break
10:45 House of Delegates Reconvenes
12:00 "Getting to Know You" CMA Luncheon, Gold Room
1:30 General Session Reconvenes
2:00 "Picking a Fight — Risk Taking"
Susan Irza, MPA, Menninger Foundation
3:00 Break, Courtesy Damon Medical Laboratories Midwest
3:15 "Guilt — It's Always Having to Say You're Sorry: Struggles of Aspiring Super Women"
Meredith Anne Titus, Ph.D., Menninger Foundation
7:30 "Stardust Banquet," Regency East
Master of Ceremonies — *William R. Allen, M.D.*
Parade of Chapter Presidents
Color Guard Presentation
Entertainment — *Rev. Keith Alberding*
Installation of Officers
Reception for Incoming Officers, Poolside Foyer, Hosted by Wyandotte County Chapter

SUNDAY, MAY 2

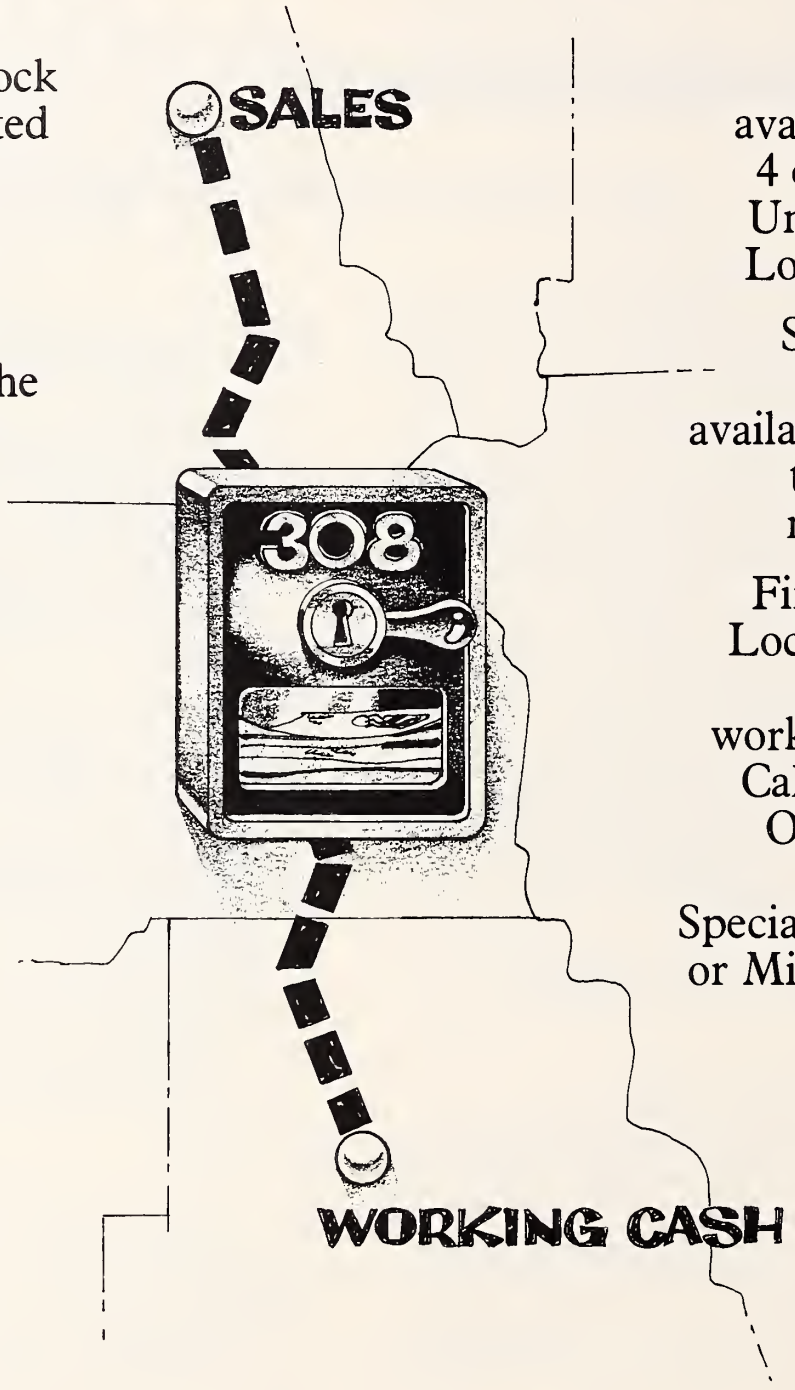
7:30 "The Lord's Prayer Breakfast," Regency West
Executive Council, Foyer East
Champagne Fountain, Courtesy of Berlin Wheeler, Inc.
Style Show
9:15 General Session, Grand Ballroom
Presiding — *Anna Marie Dercher, RT*
9:45 "Orthopedics Unusual"
Sergio Delgado, M.D.
10:45 Break
11:00 "What If . . . A Test for a Pediatric Psychologist"
Naomi Patterson, Ph.D.
12:30 "Thanks for the Memories Luncheon," Regency West
Presiding — *Anna Marie Dercher, RT*
CEUs applied for

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KaMPAC MEMBERS 1982

The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

Membership Categories

Certificate	$500—C
Sustaining	100—S
Regular	60—R

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Roger D. Warren, MD, C
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Lauren K. Welch, MD, R
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Richard E. Whitehead, MD, R

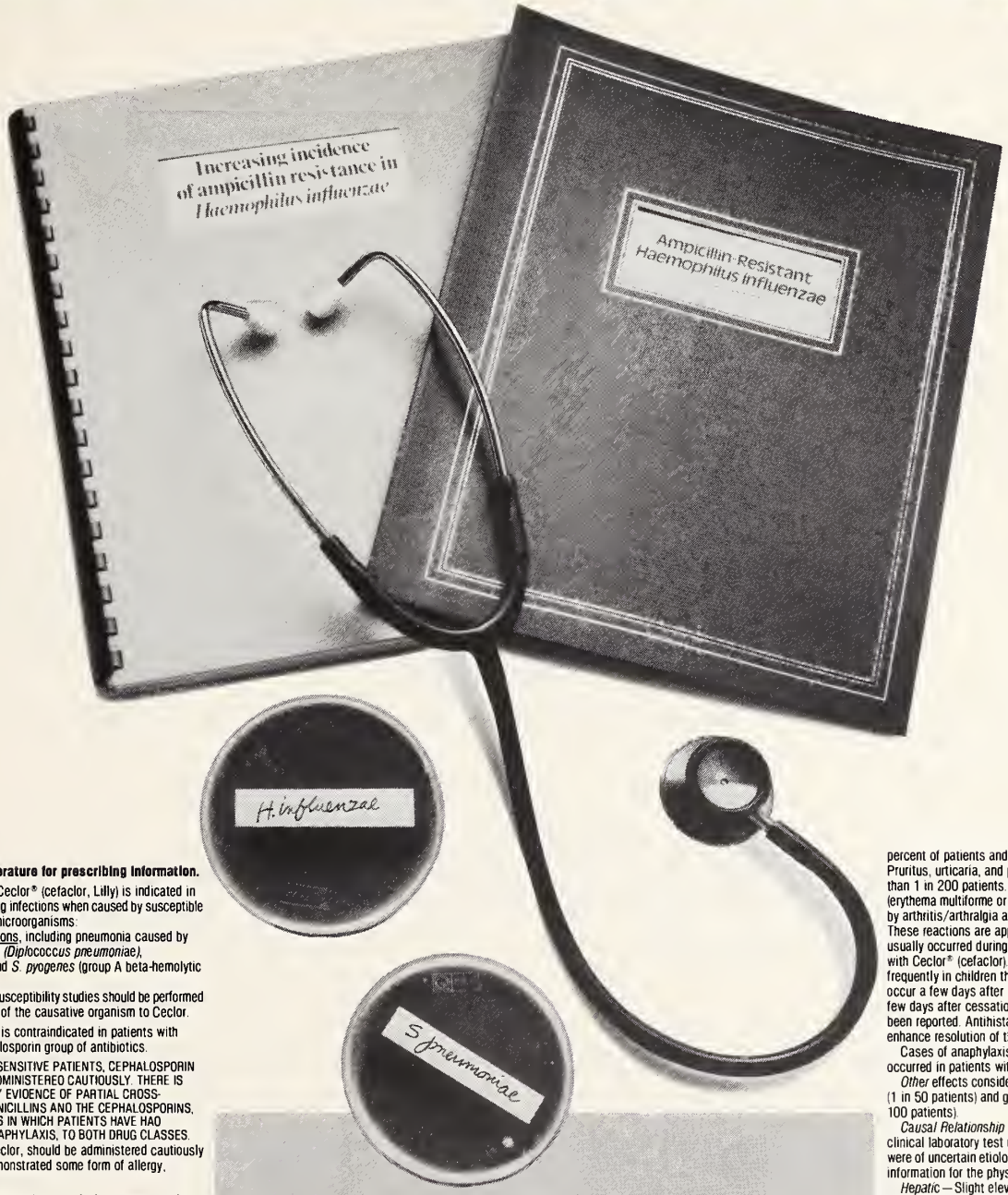
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Mrs. A. J. Wray, R
Talaat E. Yaghmour, MD, R
John W. Young, MD, R
David L. Zacharias, MD, R
David S. Zamierowski, MD, R

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Patrick N. Barker, MD, R
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A. Dean Burnett, MD, R
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Stanley W. Lowe, MD, R
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Ted L. Macy, MD, R
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Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor

Pulvules®, 250 and 500 mg

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci).

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coomb testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: Adverse effects considered related to cefaclor therapy are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70) and nausea and vomiting (1 in 90).

As with other broad-spectrum antibiotics, colitis, including rare instances of pseudomembranous colitis, has been reported in conjunction with therapy with Ceclor.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor® (cefaclor). Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, halt of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [100281R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

1. Antimicrob. Agents Chemother., *8*:91, 1975.
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7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Lilly

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J-9043-4 July 1981



In Vertigo

On Balance...

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Each Tablet Contains:
Pentylenetetrazol 25.0 mg
Pheniramine maleate 12.5 mg
Nicotinic acid 50.0 mg

Clinically proven actions

- Antihistaminic
- Cerebral stimulant
- Vasodilator

Few side effects

- Vasodilation occasionally causes facial flushing which can be minimized by recommending that Ru-Vert® be taken following meals or with food.

Dosage

- One or two tablets three times a day

Please see next page for a summary of prescribing information

MANUFACTURED & DISTRIBUTED BY

BOOTS PHARMACEUTICALS, INC.
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Pioneers in medicine for the family



In Vertigo
On Balance...
RU-VERT®

See following prescribing information.

DESCRIPTION: Each tablet contains the following active ingredients:

Pentylenetetrazol	25.0 mg
Pheniramine maleate	12.5 mg
Nicotinic acid	50.0 mg

INDICATIONS: Ru-Vert is indicated as an adjunct therapy in the symptomatic treatment of acute or chronic vertigo.

CONTRAINDICATIONS: Convulsive disorders or known history of sensitivity to any of the listed active ingredients. Because of the vasodilating action of nicotinic acid, Ru-Vert should not be used in patients with hypotension.

WARNINGS: The safety of this preparation during pregnancy and lactation has not been established. Use of this drug requires that the physician evaluate the potential benefits of the drug against any possible hazard to the mother and child.

PRECAUTIONS. Although there are no absolute contraindications to pentylenetetrazol, it should be used with caution in epileptic patients or those known to have a low convulsive threshold or a focal brain lesion. Caution should be exercised when treating patients with high doses of Ru-Vert who have heart disease. While pentylenetetrazol does not act directly on the myocardium, the results from central vagal stimulation could cause bradycardia.

Pheniramine maleate, like other antihistamines, may produce sedative side effects in certain patients.

Transient vasodilatation due to rapid absorption of nicotinic acid may produce facial flushing and a sensation of warmth. These effects may be ameliorated by recommending that Ru-Vert be taken following meals or with food.

ADVERSE REACTIONS: Pentylenetetrazol in high doses may produce toxic symptoms typical of central nervous system stimulants, which act on the higher motor centers and the spinal cord. Convulsions resulting from this drug are spontaneous and are not induced by external stimuli. They usually last for several minutes and are followed by profound depression and respiratory paralysis. Death has been reported from the ingestion of 10 grams of pentylenetetrazol.

DRUG ABUSE. Drug dependence has not been reported with Ru-Vert.

OVERDOSAGE: Signs and symptoms of acute overdose may be due primarily from overstimulation of the central nervous system and from excessive vasodilatation with resulting autonomic nervous system imbalance. The symptoms may include the following: vomiting, agitation, tremors, hyperreflexia, sweating, confusion, hallucinations, headache, hyperpyrexia, tachycardia. Treatment consists of appropriate supportive measures. If signs and symptoms are not too severe and the patient is conscious, gastric evacuation may be accomplished by induction of emesis or gastric lavage.

Intensive care must be provided to maintain adequate circulation and respiratory exchange.

DOSAGE AND ADMINISTRATION: The recommended dosage of Ru-Vert for vertigo or motion sickness is 1 or 2 tablets three times a day with meals or light snacks.

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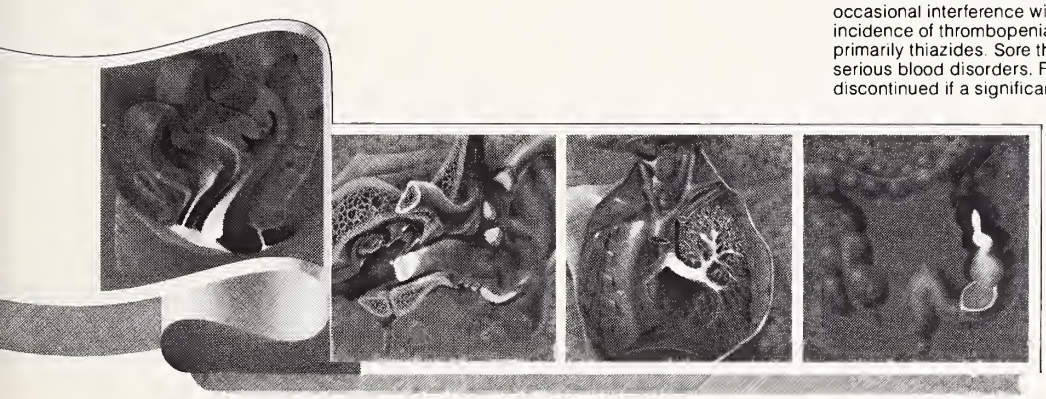
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For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. Limited clinical information presently available on effectiveness of treatment of otitis media with Bactrim when infection is due to ampicillin-resistant *Haemophilus influenzae*. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

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Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General.* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

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Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

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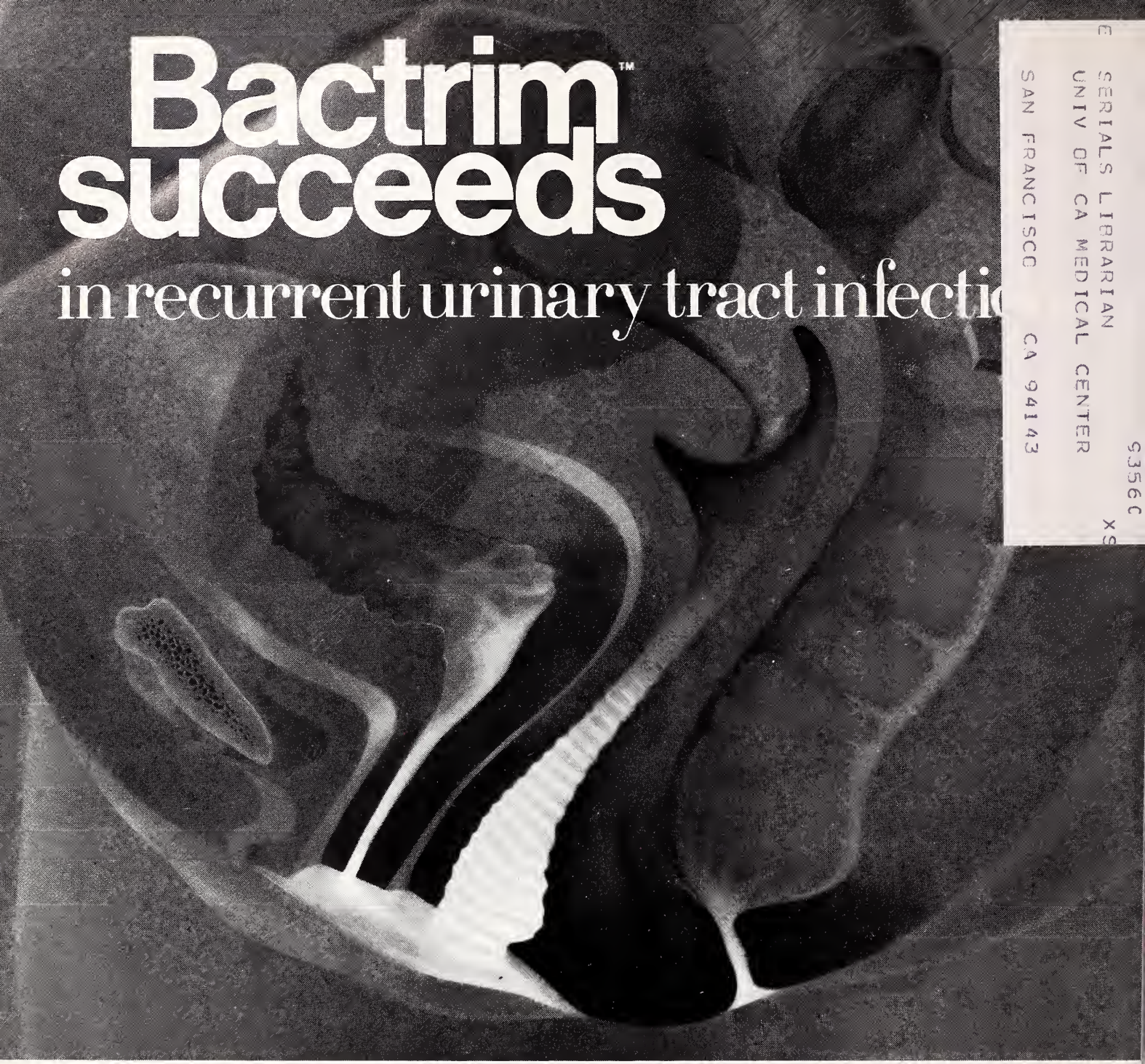
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1. Rubin RH, Swartz MN: *N Engl J Med 303*:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

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The President's Message

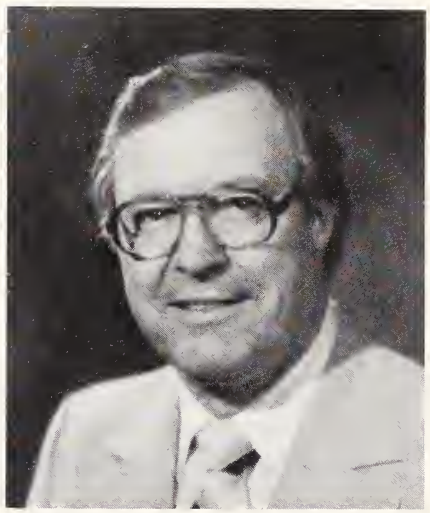
Dear Colleague:

I want to take this opportunity to thank each of you for affording me the honor of serving as President of the Kansas Medical Society. I pledge you my very best efforts and am confident that with your help, we can meet the challenges of the coming year.

The core of our organization is the Executive Staff and their ability to do the day-by-day tasks of coordinating and disseminating information to the membership. We are looking forward to working with our new Executive Director, Steve Carter, whose enthusiasm is already most evident. Jerry Slaughter will continue to serve us in a lobbying capacity. Val Braun and Gary Caruthers, with their many years of experience and expertise, are extremely essential to the various tasks as they carry out their assignments. We also recognize the importance of our membership secretary, Ramona Perez, secretaries Lee Barber and Donna Grimes, and Eleanor Bell, Editorial Assistant.

Congratulations to Herman Hiesterman for a job well done as he joins the long list of past leaders of the Society.

I also want to thank the Medical Society of Sedgwick County for the excellent manner in which they hosted the Annual Meeting this year. The scientific program, dealing with Gerontology, was both well



done and very appropriate as we face the challenge of providing care to an expanded aging patient population. We are indebted to the leadership provided by our larger county societies.

In the coming year, I hope to use this page to address some of the issues facing us in medicine. I solicit your suggestions, thoughts and criticisms by mail, telephone, or personal contact.

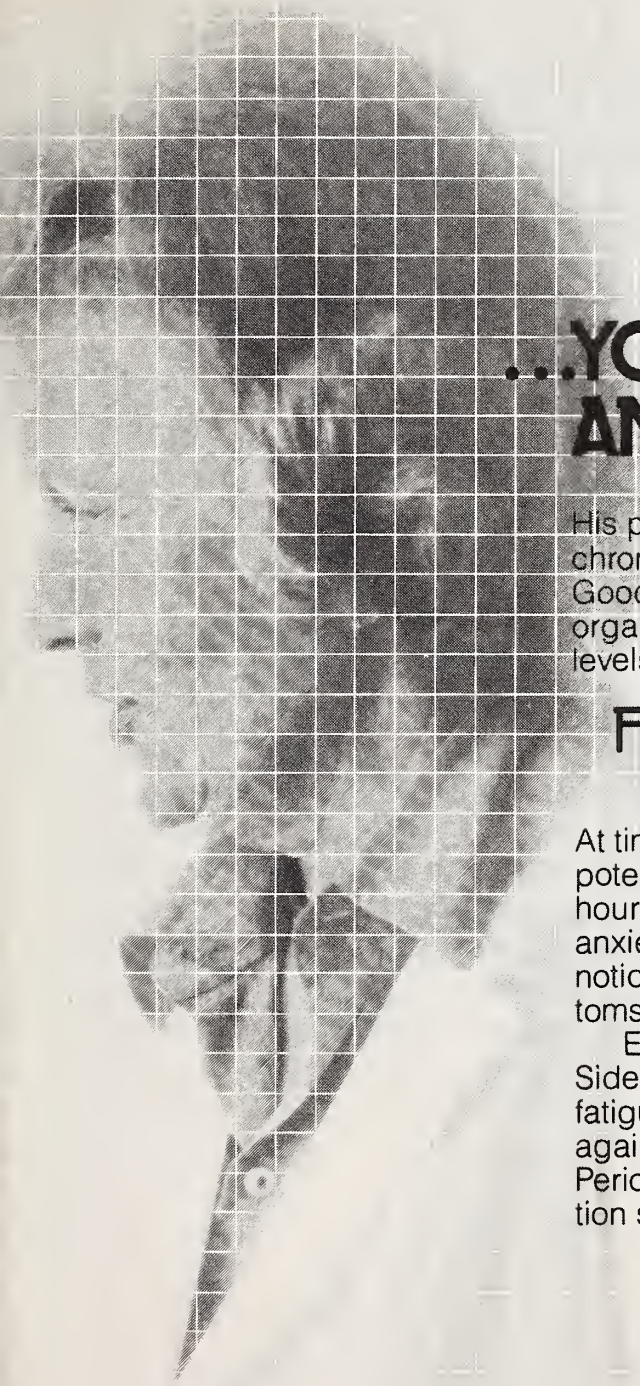
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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.
***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation. The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.
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Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
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Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

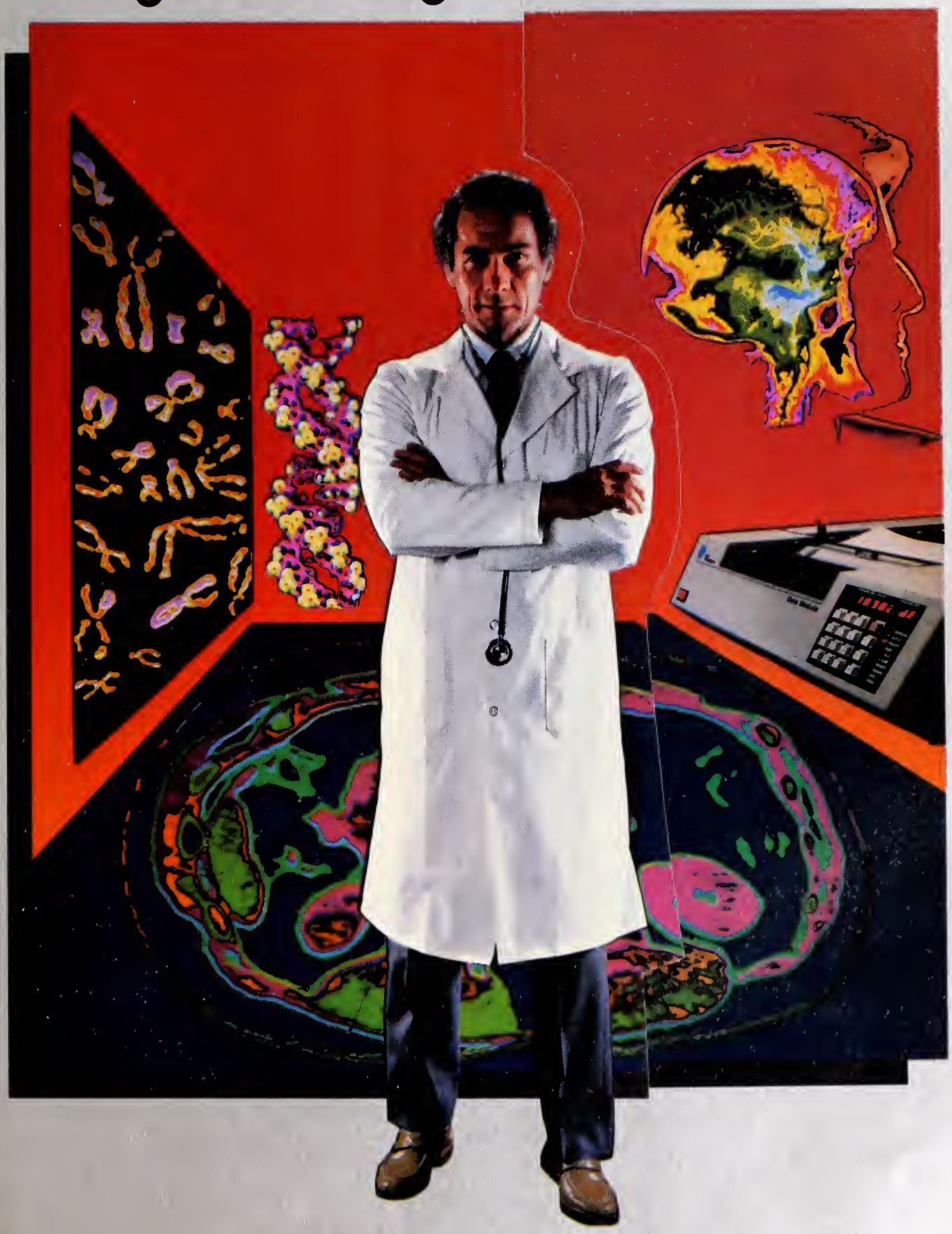
Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

In an era of change,
An Agent of Change...





Your patients are changing

. . . the population is getting older, more people are holding a second job, patients are more concerned about the medications they take.

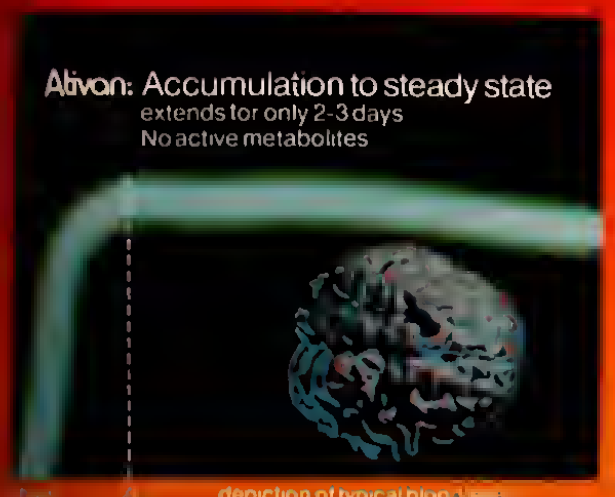
Medical knowledge is changing

. . . there are diagnostic resources and surgical techniques undreamed of only a few years ago, biomedical engineering, new insights also into the action of drugs.

In this changing environment, the way you are practicing medicine is changing too...

Twenty years ago, the benzodiazepines represented a real step forward in the management of anxiety and tension states. In recent years, however, concern about drug accumulation and clearance has led physicians to re-evaluate their use of these agents. In light of current knowledge, many clinicians are changing from multi-metabolite benzodiazepines to Ativan® (lorazepam)—a metabolically and pharmacokinetically distinctive agent that offers clinical advantages which more closely meet the expectations of a modern anxiolytic.

Ativan®: Agent of Change
(lorazepam)



depiction of typical blood





because...

it's shorter acting, with less accumulation*

In contrast to long-acting benzodiazepines, Ativan has a short, 12-hour half-life, and no active metabolites. In multiple-dose therapy, Ativan accumulates for only two to three days before reaching steady state, the long-acting benzodiazepines—diazepam CIV, chlordiazepoxide CIV, clorazepate CIV and prazepam CIV—with their active metabolites—accumulate for as long as 20 days, increasing the likelihood of excessive sedation.

it doesn't interact with drugs metabolized by P450 microsomal enzymes

Most benzodiazepines undergo oxidative metabolism and thus utilize the hepatic microsomal enzyme system. Ativan® (lorazepam), however, is metabolized by glucuronidation and does not compete with other drugs for cytochrome P450. Thus, when Ativan is given with Tagamet® (cimetidine), for example, clearance is not delayed, nor sedation increased†—unlike reported observations with patients on other benzodiazepines.[1-5]

it gives you greater control of therapy

The short half-life of Ativan® facilitates more rapid response to dosage adjustments, allowing you to titrate therapy to patients' changing needs. Also, once you decide to discontinue Ativan, it will be out of your patient's system four days after the final dose—in contrast to long-acting benzodiazepines and their active metabolites which take as long as two weeks to be totally eliminated.

Ativan®
for (lorazepam) IV
Anxiety

See important information on following page.

1 [illegible] N Engl J Med 302:1012-1014, 1980
2 De[illegible] V, Pa'vardjian RV, Schenker S, et al. Ann Intern Med 93:2[illegible]68, 1980
Pa'[illegible] RJ, Yarborough GW, Desmond PV, et al. Gastroenterology 79:912-916, 1980
4 Se[illegible]njo CA, Peachey JE. N Engl J Med 305:1255-1262, 1981
5 R[illegible] H[illegible] ompson JF, Segal JL. South Med J 74:1075-1078, 1981

*[illegible] cannot as yet be directly related to efficacy

†Although Ativan does produce additive effects when given with CNS depressants, barbiturates or alcohol



Ativan®: Agent of Change
(lorazepam) ℞



- Little accumulation lessens likelihood of excessive sedation
- Unlike most benzodiazepines, Ativan does not compete with other drugs, such as Tagamet® (cimetidine), for the microsomal enzyme system during biotransformation
- Metabolism not affected by age or liver dysfunction
- Short half-life provides greater control of therapy
- Promptly eliminated from patient's system after discontinuation
- Specifically evaluated and found effective for anxiety associated with cardiovascular and gastrointestinal disorders
- A distinctive change from long-acting benzodiazepines, all of which have active metabolites and are much the same

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions Observe usual precautions with impaired renal or hepatic function Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G I disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS Some patients have developed leukopenia; some have had elevations of LDH As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed

PREGNANCY Reproductive studies were performed in mice, rats, and 2 strains of rabbits Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses Clinical significance of these findings is not known However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide

NURSING MOTHERS. It is not known if oral lorazepam is excreted in human milk like other benzodiazepines As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%) Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken Manifestations of overdosage include somnolence, confusion and coma Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P Usefulness of dialysis has not been determined

Dosage: **Individualize for maximum beneficial effects, increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

Wyeth Laboratories Philadelphia, PA 19101

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Because as your portfolio grows, it needs managing. You can choose a brokerage firm. Or us.

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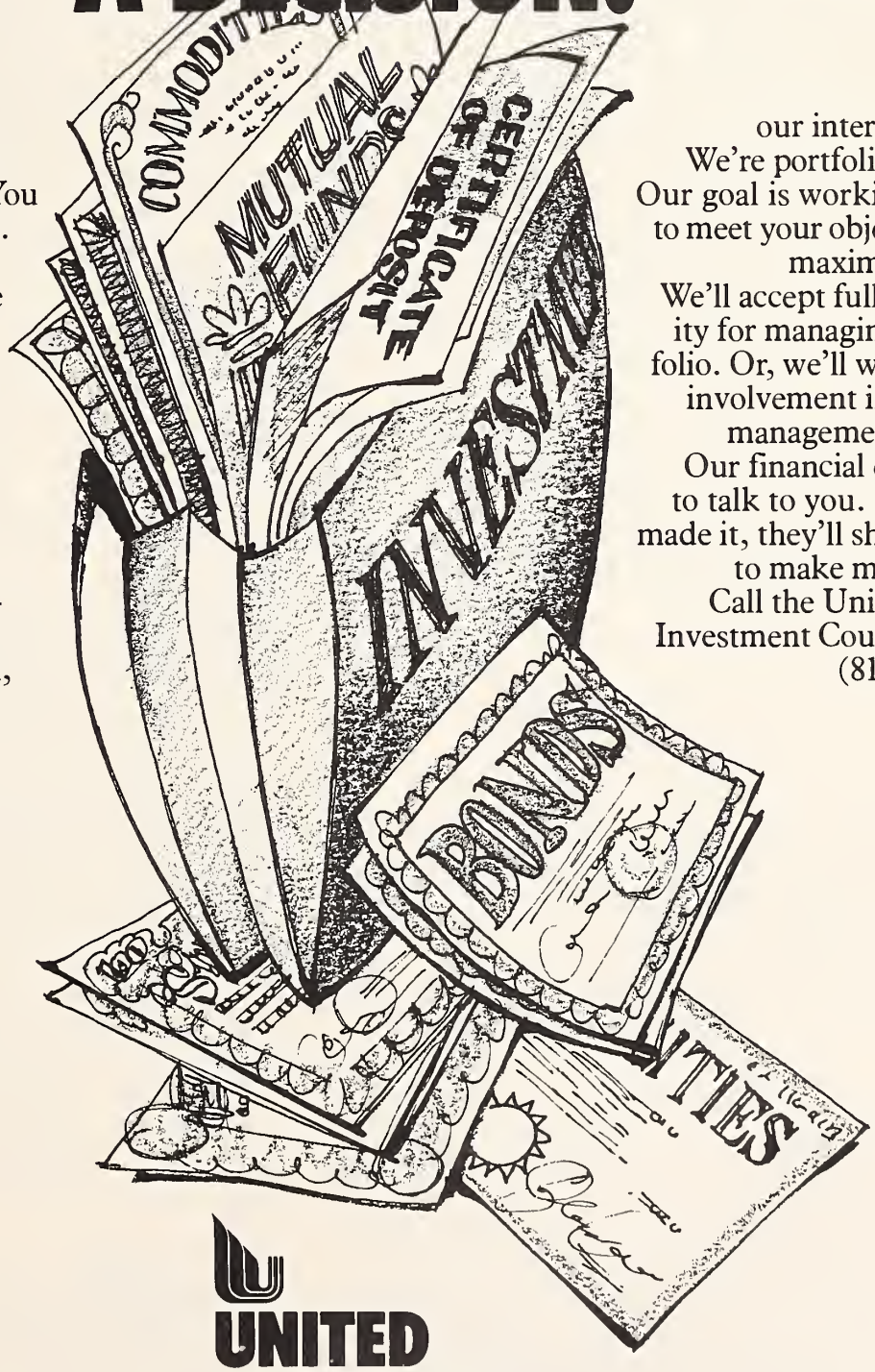
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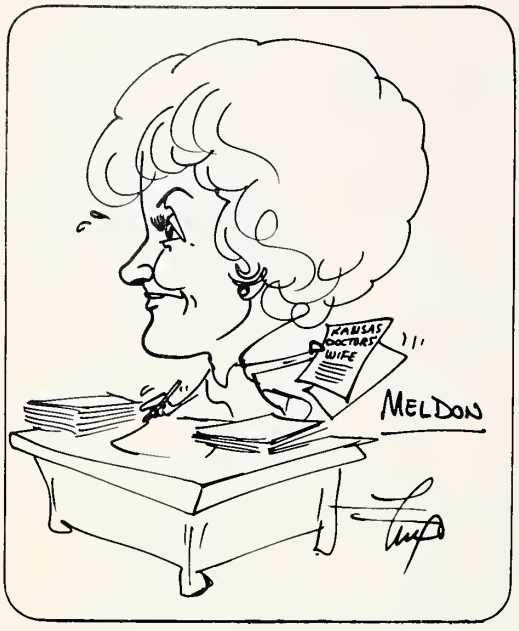


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United we grow. Together.



AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

The torch is passed . . . and I shall try to uphold the honor of my predecessors as president of your Auxiliary. I would like to tell you why 27 county presidents plus officers and chairmen — and 44 other busy Auxilians — said "yes" when asked to serve as state officers, chairmen, and committee members.

First of all, KMS Auxiliary is our link to the medical profession. Since most of us do not work in medical offices, this Auxiliary fills a need for us to be a part of the profession to which our husbands are dedicated. Physicians' wives are a privileged group — and we know it. We also know that "with privilege goes responsibility" — in this case, a responsibility to the medical profession.

KMSA is limited in membership to wives and husbands of physicians. We need *100 per cent* membership.

KMSA members are kept informed on pending legislation that affects your profession — and we write the letters to Congressmen that you don't have time to write. We are pleased that wives of three physicians are in our State Legislature (Norma Daniels, Jessie Branson, and Rochelle Chronister).

KMSA's International Health Committee sends medical supplies to Project Concern and other overseas organizations. Through the tireless leadership of Dee Cauble of Wichita and her committee, Kansas has contributed more in time and supplies than any other state.

KMSA started the "Learning Center" for fourth and fifth grade classrooms. The Center is a series of games, quizzes, and exhibits concerning pollution of the body by drugs. Instructions and kits have been sold to several other states. One Auxilian (Jean Crouch, Topeka) is trained in genetic counseling and speaks on the subject throughout the state.

KMSA raised $28,798.17 last year for medical education and research. We, of course, are a component of the AMAA, and together we raised $1.7 million last year for medical education and research — a total over the years of $31 million!

Even if we did none of the above, this organization would be worthwhile if it existed only for the purpose of promoting friendship among physicians' wives. If your wife is not a member of KMSA, urge her to join. If she is a member, ask her to become an active participant. If she is already an active participant, give her a hug!

Thank you for the financial support you give us, and for the encouragement so many of you have given to our Auxiliary. We hope the efforts of this organization will be a credit to you.

Meldon Laury
President
Kansas Medical Society Auxiliary

P.S. I have used "wife" instead of "spouse" simply because I deplore the word "spouse." We do, indeed, have three or four physicians' husbands, and we welcome them. If you are a woman physician reading the above — please substitute "husband" for "wife."

LET'S TALK BUSINESS

A continuing series on business communication issues.

Making telecommunication decisions: consider the service.

When you think about adding equipment or new systems to your business, a major consideration must be how it will be serviced, maintained and updated.

This is especially critical with today's complex and vital communications systems.

How much a system costs should only be one factor in your decision. Keeping it operating properly and efficiently is every bit as important.

Know your vendor.

Do the telecommunications vendors you're considering offer help in terms of service guarantees?

Do they provide service contracts? And if so, how much do they cost and what are the renewal terms?

Is their service reliable, fast, efficient?

Answers to questions like these should have a definite bearing on the decision you have to make.

Full risk responsibility.

When you sign a contract for service with Southwestern Bell, you're not signing for a piece of equipment, but rather for a full range of telecommunications services.

Services that might otherwise be available at a separately stated charge, or not at all.

For instance, if your Bell system should be damaged or destroyed by fire, flood, or other natural disaster, it would be replaced or repaired at no additional charge.

Our service organization is trained to respond to emergencies and handle maintenance efficiently.

In fact, by using a system we call RMATS, over 60% of the problems that develop in our larger electronic switching systems are automatically corrected before the customer is even aware the problem exists.

Systems that grow as you grow.

You're in business to grow. And business conditions are constantly changing.

Will the communications system you're considering handle your needs as your business changes?

Is your vendor prepared to work with you and make changes to meet your evolving business needs?

As technology advances, the Bell System will be designing step-by-step paths to evolve your system into the future.

Bell System consultants will be available to evaluate your needs as your business changes, to re-engineer or "re-balance" your system to match the new demands placed upon it, both from a hardware and software standpoint.

And, there is no additional cost for our consultation and evaluation.

Service means more than maintenance.

But a vendor that only provides hardware maintenance for your system is not providing full service.

Our attitude at Southwestern Bell is that we must help customers solve business problems.

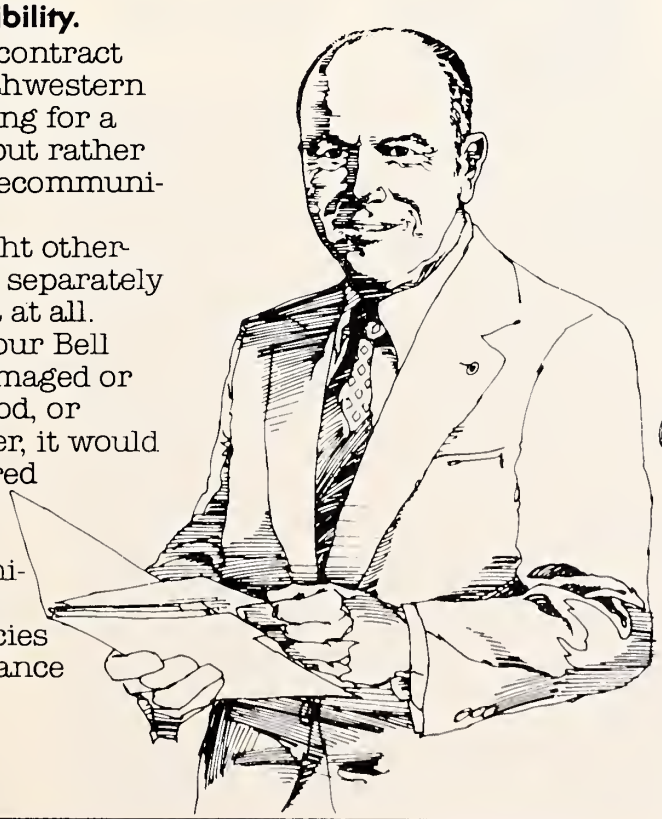
And that goes far beyond reliable equipment maintenance. It means focusing on the specific business needs of widely varied groups of customers. And providing an industry-specialized sales force that can respond with in-depth expertise.

Our business is service.

The Bell System is committed to keeping your communications service the best in the world.

We've built our reputation on service. It's something you've come to expect – even demand from our organization.

That's a solid value difficult to measure in dollars and cents. But absolutely invaluable in peace of mind.



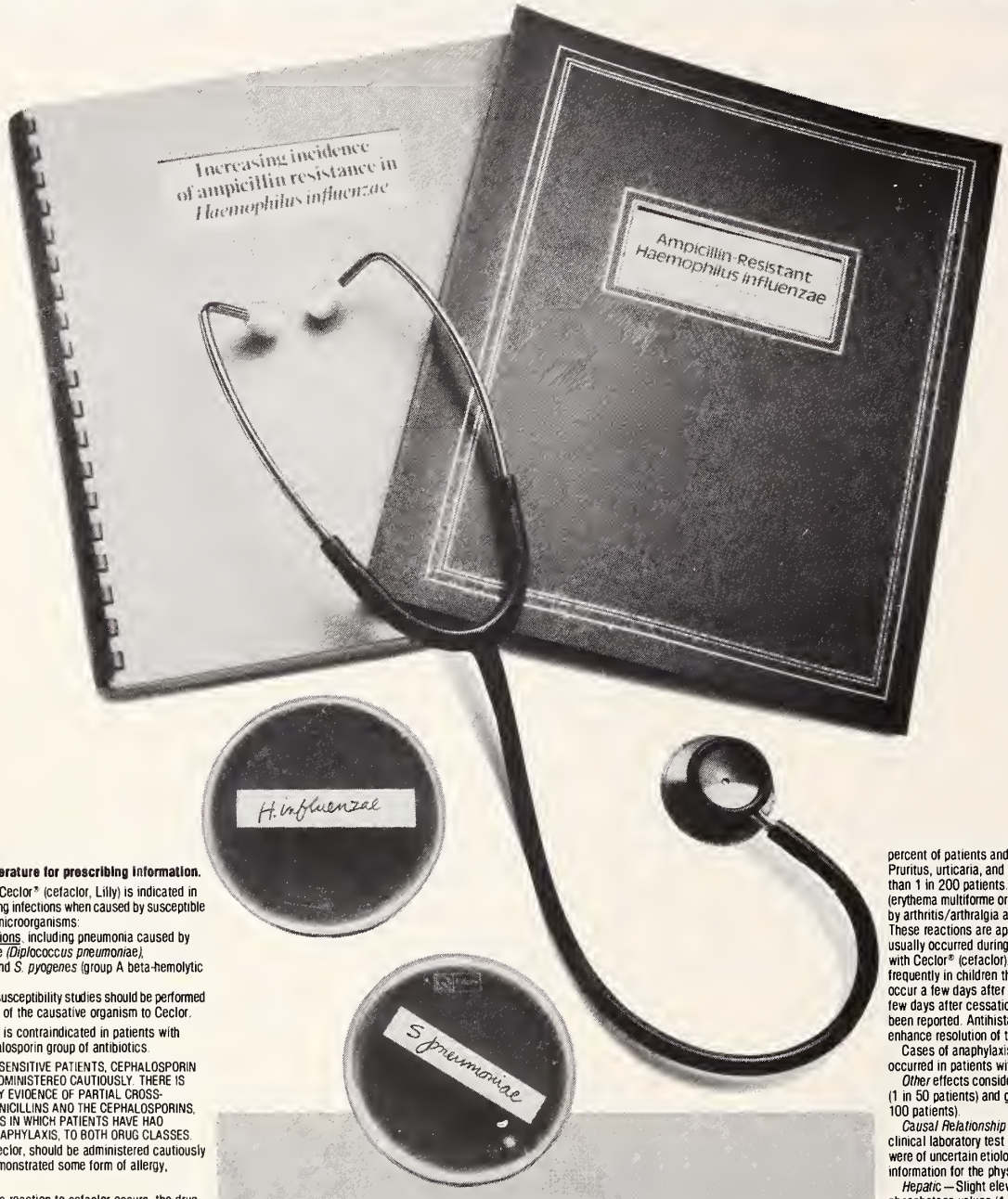
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Southwestern Bell

An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor
Pulvules®, 250 and 500 mg

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coomb testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: Adverse effects considered related to cefaclor therapy are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70) and nausea and vomiting (1 in 90).

As with other broad-spectrum antibiotics, colitis, including rare instances of pseudomembranous colitis, has been reported in conjunction with therapy with Ceclor.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor® (cefaclor). Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [100281R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

200086

KaMPAC MEMBERS 1982

The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

Membership Categories

Certificate	$500—C
Sustaining	100—S
Regular	60—R

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(Continued on page 259)



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Pheniramine maleate	12.5 mg
Nicotinic acid	50.0 mg

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WARNINGS: The safety of this preparation during pregnancy and lactation has not been established. Use of this drug requires that the physician evaluate the potential benefits of the drug against any possible hazard to the mother and child.

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Pheniramine maleate, like other antihistamines, may produce sedative side effects in certain patients.

Transient vasodilatation due to rapid absorption of nicotinic acid may produce facial flushing and a sensation of warmth. These effects may be ameliorated by recommending that Ru-Vert be taken following meals or with food.

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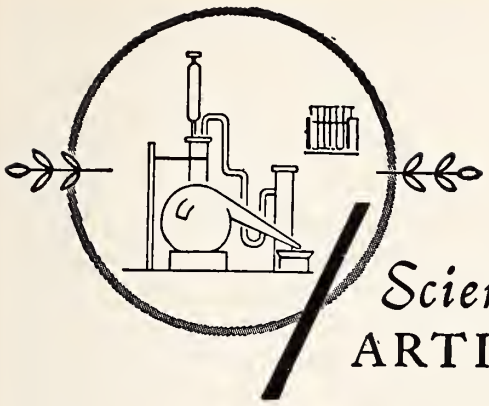
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Minor Rectal Bleeding

Treatment Guidelines for Infants and Children

TIMOTHY P. O'HOLLERAN, M.D.; LAWRENCE W. O'HOLLERAN, M.D., *and* MEDO MIRZA, M.D., *Wichita*

During the years 1970-1979, 55 pediatric patients were admitted to Wesley Medical Center for asymptomatic, minor rectal bleeding, none with significant anemia. The data on these patients suggest that routine use of barium enema and sigmoidoscopy is not warranted for infants and children in such cases.

CLINICAL RECORDS of 55 pediatric patients with asymptomatic minimal rectal bleeding were reviewed. These patients were seen at Wesley Medical Center 1970-1979. Patients' ages ranged from one day to 12 years, and all presented with a history of bright red blood per rectum, without significant physical findings. No patients presented with anemia. Duration of bleeding ranged from one episode in many patients to that of frequent, blood-tinged stools in others.

Three patients were dismissed following observation with no diagnostic procedures other than complete blood count. Fifty-two underwent barium enema, sigmoidoscopy, or both. Barium enema was utilized for 45 patients and sigmoidoscopy for 31. Seventeen patients had a Meckel's radionuclide scan.

Results

A positive diagnosis was made in only 21 of the 55 patients. Thirty-four were signed out with a notation of rectal bleeding of unknown origin. According to the records, none of these 34 patients returned with significant pathology following dismissal.

All colon x-rays were negative. Sigmoidoscopy revealed no pathology in 23 of the 31 patients thus examined. It did reveal anal fissure in two patients and juvenile polyps in six. Anal fissure was diagnosed in four additional patients by physical examination alone, for a total of six children with this diagnosis. A juvenile polyp was diagnosed as a source of bleeding in one additional patient by physical examination, and in another following examination of tissue passed per rectum prior to admission. Thus there were a total of eight patients with juvenile polyps. Of the 17 patients who underwent Meckel's radionuclide scan, none was found to have a Meckel's diverticulum as a source of bleeding. Other diagnoses assigned prior to dismissal were chronic constipation (1), enterocolitis (1), milk allergy (3), rectal prolapse (1), and rectal tag (1).

Discussion

Bleeding per rectum in the pediatric age group is relatively common. This review is limited to patients

From the Department of Surgery, Wesley Medical Center, Wichita.

Presented at the Annual Meeting of Kansas Chapter, American College of Surgeons, December, 1981.

Address reprint requests to Dr. Timothy P. O'Holleran, Wesley Medical Center, 550 No. Hillside, Wichita KS 67214.

with asymptomatic, minimal rectal bleeding. Minimal bleeding is a vague term, but none of the patients in this series presented with significant anemia requiring iron therapy or blood transfusion. Most of these cases involved blood-streaked stools, rather than gross bloody discharge. The absence of significant abdominal pain is also an important part of the history in this group of patients. Severe abdominal pain presents a different diagnostic problem than that involving these patients. Some papers have advocated use of barium enema as a first-line diagnostic test in the presence of hematochezia.[1] We believe that the data presented here, together with opinions from other authors, support the selective use of this procedure.

Gerson *et al.* have pointed out that the barium enema is relatively expensive and time consuming. The patient experiences some discomfort and radiation exposure, and performance and interpretation require a great deal of the radiologist's time.[2]

Rectal bleeding in the neonate is most commonly idiopathic without any evidence of the source after thorough investigation. The bleeding stops spontaneously. In their study of neonates with gastrointestinal bleeding, Sherman and Clatworthy were unable to reach a diagnosis in the majority of cases utilizing routine contrast radiologic examinations.[3]

Anal fissure is reported to be the most common source of minor rectal bleeding in infants and children.[4] The incidence in our series was only 11 per cent; however, the true incidence of this disorder is probably much higher because many of these patients do not present to the physician's office or hospital.

Spencer has stated that an anal fissure can be seen only when specifically looked for by spreading the perianal skin to evert the anal canal. It will not be revealed by routine examination or by proctoscopy.[4]

Meckel's diverticulum is usually seen in a child less than 3 years old with bright red, painless, rectal bleeding. Routine contrast studies are seldom helpful in making this diagnosis.[5]

Some investigators propose a barium enema for all patients with rectal bleeding in order to rule out colon polyps; yet 70 per cent of these polyps, when solitary, are within 25 cm of the anus and can be diagnosed and excised with the sigmoidoscope or colonoscope.[6] In our review, the colon x-ray failed to reveal any polyps. These juvenile polyps have no malignant potential,[6, 7] and the main reason for their removal is the presence of bleeding or obstruction.[8]

We feel that asymptomatic, minor rectal bleeding can be approached differently than profuse bleeding or bleeding associated with symptoms. The barium enema yielded negative results in all of our patients. It should be used when bleeding is major or when it is symptomatic, recurrent, or associated with anemia, but routine use of this procedure exposes patients to unnecessary expense and radiation. The sigmoidoscopic examination is also unnecessary in many cases; we believe that one or two episodes of blood-streaked stools do not warrant this procedure. We find that these problems are likely to be resolved spontaneously for patients instructed in a proper bowel training regimen. None of our patients dismissed without a definite diagnosis returned with significant pathology at a later date.

Summary

The routine use of barium enema and sigmoidoscopy is not warranted for short term, asymptomatic, minor rectal bleeding in pediatric patients. When minor bleeding is continuous or recurrent, then sigmoidoscopy is indicated. Barium enema should be reserved for patients with symptomatic, recurrent, or major rectal bleeding associated with anemia.

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Parosteal Osteosarcoma

A Case Report and Discussion

JOYCE MOORE STOVALL, M.D.; ALBERT KIM, M.D. *and* **ORLANDO J. VENTURA, M.D.,** *Leavenworth*

PAROSTEAL (JUXTACORTICAL) OSTEOSARCOMA is a rare and distinct type of osteosarcoma. It is less common than true osteosarcoma, chondrosarcoma, and even fibrosarcoma of bone.[1] This neoplasm comprises an incidence of 1.6 per cent of all proven primary malignant bone tumors and less than 5 per cent of all osteosarcomas. It has well-defined radiographical and histopathological features. Presented here is the case of a patient with parosteal osteosarcoma.

Case Report

A 24-year-old male complained of a mass on his left leg. He first noticed it two years prior to presentation and stated that it was increasing in size and was sometimes painful when he walked.

Examination revealed a hard, non-tender, irregular mass on the anterior aspect of the middle third of the left tibia.

Radiographs of the left lower leg revealed a densely ossified mass that was clearly defined with periosteal elevation at the medial aspect of the mid tibia *(Figure 1)*. The lateral view suggested cortical thickening of the medial aspect of the mid tibia *(Figure 2)*. There was a radiolucent line of demarcation between the dense tumor and the cortex that was seen on tomograms without apparent bone destruction *(Figure 3)*. A radioisotope bone study demonstrated a single area of increased tracer accumulation involving the left tibia corresponding to the abnormality seen on the radiograph *(Figure 4)*. Results of the chest radiograph and laboratory studies were normal.

The mass in the left leg was excised and this revealed low grade osteosarcoma most compatible with that of parosteal osteosarcoma.

Clinical Findings

Parosteal osteosarcoma usually develops very slowly by progressive proliferation of periosteum. The age range is from childhood to 60 years with 50 per cent of cases presenting in persons aged 30 years and older. There is no sex predilection; males and females are equally affected. The patient frequently presents with a small or large mass on the surface of a limb bone. The tumor is usually asymptomatic, and if pain does occur, it is a late feature which is dull and intermittent. The tumor that is situated superfically is comparatively painless in contrast to a deep seated lesion.[2] If the neoplasm is located near a joint, it may interfere with articular motion.

Parosteal (juxtacortical) osteosarcoma is a malignant bone neoplasm that usually has a predilection for the metaphyseal end of the femur. A patient presented complaining of a mass on the middle third of the tibia. A biopsy of the tumor revealed parosteal osteosarcoma. The clinical, radiological, and histopathological findings are reviewed.

Two distinct types of parosteal osteosarcoma have been described by Lorentzon *et al.*[3] The predominant type is originally a benign, slow-growing tumor but has a definite malignant potential, and metastases may occur after long symptom-free intervals. A radical en bloc excision of the tumor, including all of the surrounding soft tissues and affected muscles or fascia, followed by regular review is recommended. The other type is initially a rapid growing, highly malignant tumor, and primary amputation is recommended. Parosteal (juxtacortical) osteosarcoma may metastasize by way of the bloodstream to the lungs, particularly after attempts at local excision.[4]

Radiological Findings

The neoplasm almost always begins at the metaphyseal end of an affected tubular bone and has a sessile or broad base. The distal femur is predominately involved, although occasionally the tibia, humerus, ulna, or some other long bone may be affected.

From the Radiology and Pathology Services, Veterans Administration Medical Center, Leavenworth.

Address reprint requests to Dr. Stovall, V.A. Medical Center, Leavenworth, KS 66048.



Figure 1. An anterior posterior view showing new bone formation and periosteal elevation of the medial aspect of the mid tibia.

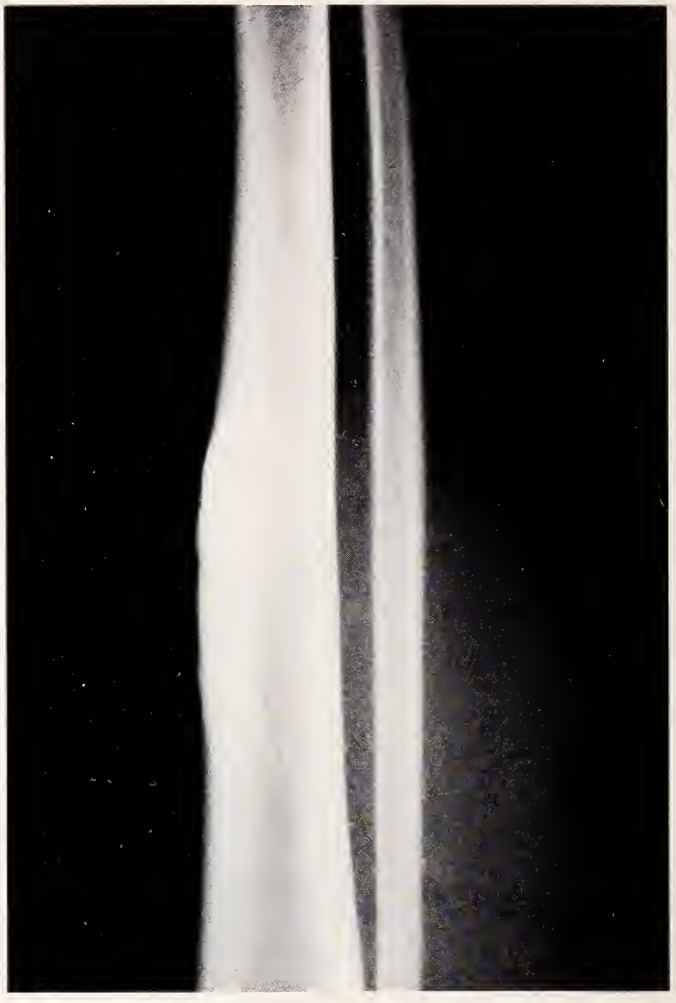


Figure 2. A lateral view of the mid third of the left tibia suggests some thickening of the cortex along the medial aspect.

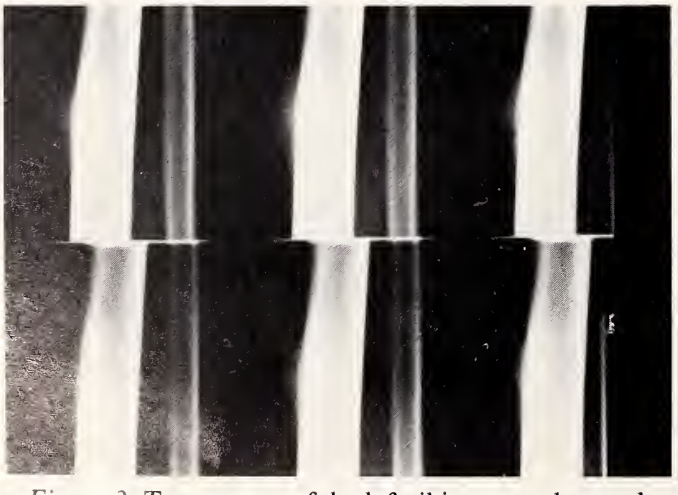


Figure 3. Tomograms of the left tibia were taken and a radiolucent line of demarcation between the dense tumor and the cortex is seen without apparent bone destruction.

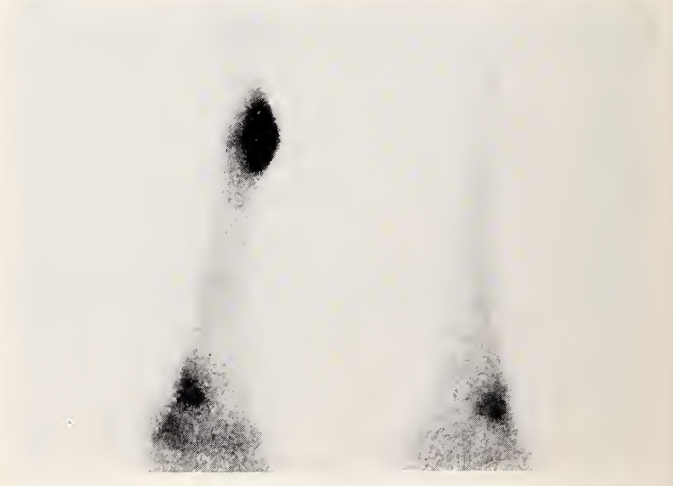


Figure 4. A radioisotope study demonstrated an increased area of uptake of the tracer in the mid portion of the left tibia.

In the early stage there is a narrow radiolucent zone of demarcation that separates the dense tumor from the cortex. Periosteal elevation may be visible in the early stage. If left untreated, the neoplasm extends circumferentially and longitudinally to surround a major segment of bone with or without invasion.[5] The malignant type tumor invades the cortex and spongiosa, producing areas of lysis interspersed with new bone formation,[2] which may also be present in soft tissue or other bones.

Arteriography may be helpful in that, if abnormal vessels are present, the tumor is almost always actively malignant. Computerized tomography may play an important role in helping to evaluate this neoplasm and other bone tumors by offering a unique cross-sectional display which may clearly demonstrate whether there is cortical or medullary bone involvement or both, as well as soft tissue involvement. CT scans may permit possible detection of skip metastases proximal to the lesion in long bones or in adjacent soft tissue.[6]

The differential diagnoses include post-traumatic myositis ossificans, osteochondroma, extraosseous osteosarcoma, fracture callus, ossifying hematoma, osteosarcoma of soft tissue, osteosarcoma of bone, pseudo-malignant osseous tumor of soft tissue, periosteal osteogenic sarcoma, and chondrosarcoma.

Histopathological Findings

Parosteal osteosarcoma is often difficult to identify, particularly from a biopsy specimen, because there may be zones in which the bone and osteoid are well oriented, closely resembling normal bone, such as in our case. The osteoblasts show little indication of their neoplastic character, and the intervening fibrous stroma is rather well differentiated and moderately cellular. The nuclei exhibit little or no pleomorphism. Some lesions have more histological indications of malignancy with fairly cellular stroma, neoplastic osteoid, chondroid, or a combination of these components.

Of the mentioned differential diagnoses, osteochondroma, post-traumatic myositis ossificans, and extraosseous osteosarcoma may present diagnostic problems. In osteochondroma, stromal changes such as hypercellularity are lacking and islands of cartilage will be seen in the bony trabeculae.[7] In myositis ossificians, the zoning effect is identified in the biopsy specimen. Extraosseous osteosarcoma is less likely to be as well differentiated as parosteal osteosarcoma.

It is often difficult to make a diagnosis and it is sometimes quite helpful for the pathologist to review the case with the radiologist and orthopedist.

Acknowledgement

Adres Grisolia was the orthopedist in the case reported. Philip E. Koontz, Library Service, retrieved the literature for this paper.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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Tissue Necrosis

Cutaneous and Subcutaneous Damage Following Extravasation of Methylene Blue

JAMES L. RUHLEN, M.D, *Olathe*

TISSUE NECROSIS is among the adverse effects of methylene blue injection listed by the American Hospital Formulary, but no references are provided. In 1974, Perry and Meinhard reported necrotic subcutaneous abscesses of the distal foot following injection of methylene blue for lymph node marking.[1] Necrosis of the spinal cord following intrathecal injection of methylene blue has been reported.[2] Methylene blue is still used intravenously in the treatment of drug-induced methemoglobinemia, and as a tissue marker in some surgical applications.[3] Necrosis of subcutaneous tissues following methylene blue injection has not been previously reported in the American literature. This report describes a patient who developed serious tissue necrosis following attempted intravenous injection of methylene blue.

Case Report

A 35-year-old male was brought to the emergency room after being found unresponsive by friends. The emergency room physician noted profound cyanosis of the nailbeds and lips, unresponsiveness to verbal commands, and minimal responsiveness to pain, in a thrashing and apparently combative patient. Initial blood pressure was reported to be 100 mmHg systolic by palpation only. Suspecting methemoglobinemia, the emergency room physician injected methylene blue, 70 mg, through an intravenous line which had been established in the left antecubital fossa. Shortly thereafter, the intravenous line was noted to be infiltrated, and was discontinued. The patient showed no improvement, and was subsequently found to be in profound vascular collapse and shock, due to the effects of an acute gastroenteritis on previously unsuspected severe adrenal insufficiency and diabetes insipidus, secondary to remote head trauma. He was resuscitated with some difficulty, utilizing massive intravenous fluid replacement, ventilatory support, dopamine, and high-dose intravenous corticosteroids. No dopamine was administered into the left arm. Within 90 minutes after methylene blue injection, a 3 x 3 cm zone of profound skin pallor, with surrounding traces of blue color, was noted at the site of injection. During the next 48 hours, frank necrosis of the initially pale skin developed, and during the next five days, the area of necrosis enlarged to approximately 6 x 8 cm. An incomplete left median nerve palsy developed. On the eighth hospital day, a plastic surgeon performed a debridement procedure. He found complete necrosis of the skin, subcutaneous tissue, and underlying fatty tissue of the left antecubital fossa. When the superficial fascia of the arm was excised, dark blue coloration oozed into the wound. The biceps muscle had viability; however, bleeding was not very brisk in all areas. Necrotic tissue and blue dye were cleansed as completely as possible, and a split-thickness skin graft was obtained from the left thigh and applied over the defect. The patient was dismissed from the hospital the following day on intranasal desmopressin, 0.1 cc two times per day; prednisone, 10 mg two times per day; levothyroxine, 0.3 mg/day; and fluoxymesterone, 10 mg/day.

Although necrosis of subcutaneous tissue is among possible adverse effects of methylene blue injection, this problem has not been previously reported in the American literature. The case here reported demonstrates the possible serious consequences of extravasation.

Approximately one week later, the skin graft sloughed, and the patient required further plastic surgery, utilizing a tissue flap taken from the upper arm. Eighteen months later, the patient had a slight residual left median nerve palsy, incomplete extension of the left arm at the elbow, and diminished muscle mass of the left forearm and hand.

Discussion

This patient suffered accidental injection of methylene blue into the subcutaneous tissues of the left antecubital fossa, as evidenced by the observed

(Continued on page 260)

Address reprint requests to Dr. Ruhlen, 807 So. Clairborne, Olathe KS 66062.

Metastatic Malignant Melanoma

Symmetrical Involvement of Both Ureters

GREGORY C. DOWNING, M.D. *and* JAMES A. WHITAKER, M.D., *Wichita*

Metastatic malignant melanoma involving the genitourinary tract is becoming more frequently recognized. Ureteral implants are less common than are renal or bladder metastases. Described here is an unusual case of symmetrical ureteral metastases which presented in an atypical fashion.

Case Report

A 34-year-old white male presented with a three-day history of rapidly progressive memory loss. An emergency CT brain scan showed partially calcified, multiple intracerebral metastases. The patient was therefore admitted to search for a primary malignancy.

Initial physical examination showed disorientation as to person, place, and time. An enlarged axillary lymph node was discovered and biopsied. Pertinent laboratory data included an hemoccult positive stool test, and hematuria with highly atypical cells in urinary sediment. Results of chest x-ray were negative. Excretory urography demonstrated bilaterally symmetric filling defects at the pelvic inlet *(Figure 1)*. A gallium scan was consistent with disseminated malignant disease. The patient died on the 17th hospital day. An autopsy disclosed widespread malignant melanoma involving the gastrointestinal and genitourinary tracts as well as the thyroid, adrenals, and brain. The previously biopsied axillary lymph node was also involved. Specifically, the ureteral filling defects were found to be intraluminal malignant melanoma nodules *(Figure 2)*.

Discussion

Urinary tract involvement by metastatic melanoma is often clinically silent and usually indicates a poor prognosis. Painless hematuria may be the only presenting symptom. Although not pathognomonic, melanuria is a good indicator of genitourinary involvement. Das Gupta reported 45 per cent renal involvement and 18 per cent bladder involvement with metastases to other organs of the urinary tract occurring rarely.[1] Ureteral metastatic melanoma was found in five of 71 patients by Goldstein.[2] At least two of these five patients had bilateral involvement either as non-obstructing lesions at the ureteropelvic junctions or as multiple filling defects in the pelvis and ureters.

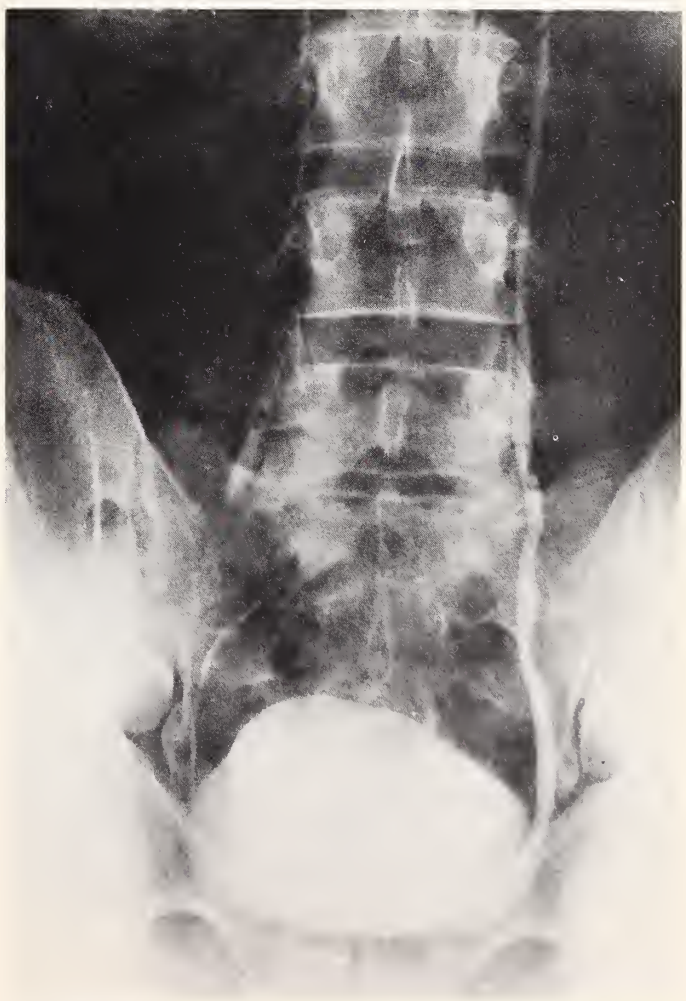


Figure 1. Excretory urography demonstrated bilateral filling defects at the pelvic inlet. a. Ten minutes PA view.

From the Department of Radiology, Wesley Medical Center, Wichita.





Figure 1, b. & c. Fluoroscopic spot views of right and left ureters.

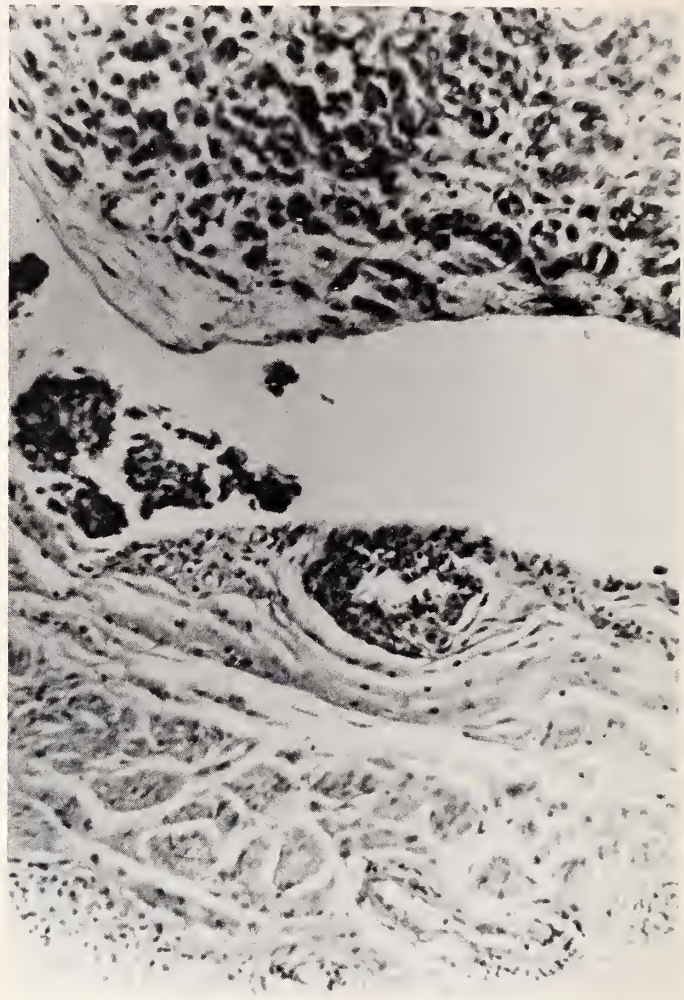


Figure 2. Microscopic section through ureteral lesion showing intramural metastasis (upper part) with intraluminal component.

This case was typical in most respects. It was atypical in its radiographic presentation, *i.e.*, symmetrical filling defects at the pelvic inlet on intravenous pyelogram. This has not been reported in the literature we have reviewed. Metastatic malignant melanoma should always be considered if neoplasia of the genitourinary tract is suspected. Further, in cases of proven malignant melanoma, excretory urography should be an integral part of the patient's evaluation.

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Vulvar Carcinoma

A Case of DDP-Induced Neurotoxicity in Chemotherapy

MARTHA HURLEY, M.D.; RONALD L. STEPHENS, M.D.; MARK L. DAVIDNER, M.D. *and* **JAVIER F. MAGRINA, M.D.,** *Kansas City, Kansas*

A woman with basalosquamous carcinoma was treated with several consecutive therapy regimens, including cis-diaminedichloroplatinum (DDP) chemotherapy. She subsequently developed various commonly recognized side effects,[1] as well as "stocking-glove" peripheral neuropathy, of which there is only one reported case in the literature.[2] Findings implicate DDP as the cause, adding to the list of toxicities to be monitored in DDP chemotherapy.

CIS-DIAMINEDICHLOROPLATINUM (DDP) is a relatively new antitumor agent which has been shown to be effective in the treatment of a wide variety of tumors. Major side effects include renal, audiologic, hematologic, and gastrointestinal complications. In this report, a woman with vulvar carcinoma was treated with DDP chemotherapy after failure to achieve complete remission with surgery and radiotherapy, and she subsequently developed a peripheral neuropathy.

Case History

A 33-year-old white female was diagnosed in June 1967 as having basalosquamous carcinoma of the vulva; it was treated by local excision. She had a recurrence of the cancer in July 1970, and underwent a simple vulvectomy in August. She had an uneventful pregnancy and an uncomplicated delivery in 1974. In 1976, she developed palpable left groin nodes. A left inguinal node dissection revealed metastatic basalosquamous carcinoma to multiple groin nodes. Between February and June 1977, the patient received a maximum dose of cobalt therapy to the pelvis. Two new lesions appeared on the labia, and one in the left femoral triangle during this time.

In June 1977, she was started on cis-diaminedichloroplatinum, and received 50 mg/M^2 for five days.

Following administration of the first course, she developed tinnitus and a moderately severe high frequency sensori-neural hearing loss. Following resolution of ototoxicity, she received six additional courses of DDP, 100 mg/M^2 per course, at three week intervals. DDP therapy was then discontinued because creatinine clearance had dropped from 64 to 36 ml/min.

In October 1977, during DDP therapy, the patient noticed tingling and paresthesias bilaterally in hands and feet. She experienced increasing dysesthesias during a three-month period, lost some function in her fingers, and stated that her mental function had deteriorated. Neurological examination revealed bilateral asymmetric weakness of grip and finger flexors. Fine motor control was within normal limits. Mild abnormalities could be detected in light touch, pain, vibratory, and heat sensation. Tinel's sign was negative on the left and positive on the right. Deep tendon reflexes were absent throughout. Cerebellar function was within normal limits. Chest x-ray, bone scan, pelvic sonography, blood cell counts, and chemistry profile revealed no evidence of recurrence of the tumor. Nerve biopsy and electromyographic studies were refused by the patient. Although objective evidence is therefore lacking, clinical findings were characteristic of peripheral neuropathy. Physical examination in August 1978 showed that the neuropathy in the upper extremities had essentially disappeared; however, there was residual numbness in the finger joints, elbows, shoulders, knees, and toes.

Discussion

Cis-diaminedichloroplatinum (DDP), a heavy metal coordination compound, is an organic complex formed by a central platinum atom surrounded by chlorine and ammonia groups in the "cis" position.[3] The two labile CL^- atoms in the "cis" position are similar to the mustard-type bifunctional alkylating agents. The major toxic effects of DDP are gastrointestinal, renal, audiologic, and hematologic. In our patient, all of these toxicities were present.

Ototoxicity is found in approximately 30 per cent

of cases treated with initial doses of 50 mg/M^2 [4] and is manifested by tinnitus, hearing loss, or both. No vestibular symptoms have been reported. Ototoxicity may occur without other toxicities, and may be unilateral.[5]

The limiting factor of the therapeutic usefulness of DDP is nephrotoxicity.[6] DDP is associated with acute tubular necrosis[7] clinically evidenced by azotemia, decreased creatinine clearance, and occasionally hyperuricemia. The severity of the toxicity depends on the glomerular filtration rate. It is dose-related and cumulative, as established by Leonard in 1971.[8] Creatinine clearance of less than 50 ml/min is considered a contraindication for therapy.

Metals in close proximity to platinum on the periodic table, such as mercury and thallium, show similar toxic effects. Poisoning by these heavy metal compounds causes renal and vascular damage, gastrointestinal disturbances and neurotoxicities — including well documented central nervous system damage and peripheral neuropathies.[9, 10]

Our patient experienced several of the toxicities attributed to cis-diaminedichloroplatinum chemotherapy. She experienced nausea and vomiting following each dose. She had acute tinnitus and a residual high-frequency hearing loss. Her creatinine clearance dropped from 64 to 36 ml/min, evidence of renal impairment. This returned to normal range during the six months following therapy. Neurologically, she experienced paresthesias which progressed to a "stocking-glove" type peripheral neuropathy, and she also complained of some vague CNS disturbances. The neuropathy resolved during the year following treatment, with no significant long-term complications.

With the close relationship of platinum to mercury and thallium, it is not unexpected to see a similar picture in the neurotoxic effects of platinum compounds. There are several reports of neurotoxicity of platinum with reference to paresthesias and seizures in older patients.[11] There is one case report of a 19-year-old woman with osteosarcoma,[2] treated with DDP, who subsequently developed a peripheral neuropathy very similar to the patient's neuropathy in this case. Her "stocking-glove" neuropathy improved when the DDP was held, and worsened when therapy was reinstituted.

Although renal impairment, other drugs, or tumor itself must be considered in the etiology of the disorder, the relationship of DDP administration and the development of a neuropathy consistent with the effects of other heavy metals indicates that DDP may be the causative agent in this peripheral neuropathy.

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Renal Scan

Determination of Renal Function Following Left Renal Vein Ligation

J. E. BOSILJEVAC, M.D.,* ***Emporia, and*** **S. JIM FARHA, M.D.,†** ***Wichita***

LIGATION of the left renal vein is sometimes necessary to gain additional exposure during surgery of the abdominal aorta. This ligation is well tolerated clinically if it is carried out close to the inferior vena cava, preserving collateral adrenal, gonadal, lumbar, and ureteral vessels which empty directly into the left renal vein. Renal function following renal vein ligation has been reported in the past,[1-7] but in these studies some data were obtained in the late postoperative period. In the present series, renal function was studied in the immediate postoperative period in patients who underwent left renal vein ligation during surgery for abdominal aortic aneurysm. Furthermore, information was obtained using renal scans, which appear to be more sensitive indicators of changes in renal function.

Material and Methods

Seventeen patients who had the left renal vein ligated during abdominal aortic aneurysmectomy were studied. In these patients, the left renal vein was ligated for exposure at the discretion of the operating surgeon. During the same time period, surgery was performed on 161 patients with abdominal aortic aneurysms (a 9.5% incidence of ligation of the left renal vein). Pre- and postoperative studies included blood urea nitrogen (BUN) and serum creatinine (CR), urinalysis (UA), two-hour creatinine clearance (CrCl), and intravenous pyelograms (IVP). In addition, renal scans were obtained using Tc^{99m} diethylenetriamine penta-acetic acid (DTPA) and I^{131} iodohippurate (hippuran) as renal scanning agents. DTPA demonstrates the vascular or glomerular phase, and the hippuran demonstrates the excretory or tubular phase of renal function. These agents are also useful to assess regional distribution in the functioning parenchyma. Postoperative data were obtained during the first week following surgery, depending upon the clinical course of the patient. If renal function was found to be decreased, renal scans and IVPs were obtained as an outpatient procedure until these studies were normal.

A small number of patients who had the left renal vein ligated during surgery for abdominal aortic aneurysm demonstrated a temporary decrease in left renal function on renal scan. This decrease was not demonstrated on other renal function studies, the procedure was well tolerated, and function soon returned to normal.

Results

Patients were divided into three groups. *Table I* lists data for patients in whom no change in renal function could be demonstrated by any of the studies during the postoperative period. *Table II* demonstrates findings in patients who had some decrease in left renal function postoperatively. *Table III* contains data on two patients with ruptured aneurysms. Both presented with hypotension but were not in shock, and preoperative studies are incomplete since urgent operation was indicated.

Discussion

As demonstrated in this study, left renal vein ligation during surgery for abdominal aortic aneurysm is well tolerated clinically. Use of the renal scan in the immediate postoperative period was useful in depicting slight decreases in left renal function which were not identified by other methods. The majority of patients (65%) in this study did not show any change in renal function in any of the laboratory studies, including renal scans. A small number of patients (35%) did show a decrease or no initial function of the left kidney on the postoperative renal scan, but these scans all returned to normal within two months. No clinical criteria, such as hypertension, fever, or uremia suggested that impairment of function of the left kidney was present. The two patients in this study who underwent opera-

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TABLE I

DATA FOR TEN PATIENTS (65%) HAVING NO DETECTABLE ALTERATION IN RENAL FUNCTION

	Preop.						Postop.					
Patient	*UA*	*BUN (mg/ 100 ml)*	*CR (mg/ 100 ml)*	*CrCl (ml/min)*	*Renal IVP†*	*Scan†*	*UA*	*BUN (mg/ 100 ml)*	*CR (mg/ 100 ml)*	*Renal (ml/min)*	*IVP†*	*Scan†*
1	NL	20	1.0	96	NL	NL	NL	10	1.0	90	NL	NL
2	NL	17	1.2	120	NL	NL	NL	18	1.1	113	NL	NL
3	NL	27	1.6	112	NL	NL	NL	25	1.5	111	NL	NL
4	NL	17	1.1	84	NL	NL	NL	12	1.1	81	NL	NL
5	NL	18	1.1	134	NL	NL	1+ Protein	20	1.1	129	NL	NL
* 6	NL	22	1.2	—	—	NL	NL	20	1.2	—	—	NL
* 7	NL	14	1.3	—	NL	NL	NL	12	1.2	—	—	NL
* 8	1+ Protein	15	1.0	—	—	NL	NL	13	1.0	—	NL	NL
* 9	NL	17	1.1	—	—	NL	NL	18	1.1	—	—	NL
*10	NL	19	1.2	—	—	NL	NL	19	1.2	—	—	NL

* All data not collectable in these cases.

† Changes not pertinent to this study, such as bilateral decreased function (glomerulonephrosclerosis), are excluded.

TABLE II

DATA IN PATIENTS DEMONSTRATING ALTERED RENAL FUNCTION

	Preop.						Postop.						
Patient	*UA*	*BUN (mg/ 100 ml)*	*CR (mg/ 100 ml)*	*CrCl (ml/min)*	*IVP**	*Scan**	*UA*	*BUN (mg/ 100 ml)*	*CR (mg/ 100 ml)*	*CrCl (ml/min)*	*IVP**	*Scan**	*Comment*
1	NL	11	1.1	60	NL	NL	NL	15	1.1	64	↓ Function Left Kidney	No Function Left Kidney	Scan and IVP NL 2 mos
2	1+ Protein	28	1.4	81	NL	NL	3+ Protein	25	1.3	74	NL	↓ Function Left Kidney	Scan NL in 7 day
3	NL	18	1.2	54	NL	NL	NL	22	1.4	36	↓ Function Left Kidney	Markedly ↓ Function Left Kidney	Scan and IVP NL 2 mos
4	NL	19	1.3	94	NL	NL	Trace Protein	11	1.4	104	NL	↓ Function Left Kidney	Scan NL at 2 mos
5	NL	15	1.3	89	NL	NL	NL	21	1.3	84	NL	↓ Function Left Kidney	Scan NL at 2 mos

*Changes not pertinent to this study, such as bilateral decreased function (glomerulonephrosclerosis), are excluded.

TABLE III
DATA FOR PATIENTS WITH RUPTURED ANEURYSMS

	Preop.						Postop.					
Patient	UA	BUN (mg/ 100 ml)	CR (mg/ 100 ml)	CrCl (ml/min)	IVP	Scan	UA	BUN (mg/ 100 ml)	CR (mg/ 100 ml)	CrCl (ml/min)	IVP	Scan
1	NL	—	—	—	—	—	NL	27	1.2	113	NL	NL
2	NL	12	0.7	—	—	—	NL	19	0.7	144	NL	↓ Function (NL at 2 mos)

tion for ruptured aneurysms were not in shock, and tolerated ligation of the left renal vein well.

Renal scans appear to be a sensitive indicator of slight decreases in renal function following left renal vein ligation. This sensitivity was not seen in the other renal function tests which were obtained. The IVP did show a decrease in left renal function in two patients who had *markedly* diminished function on renal scan *(Table II)*. On the other hand, slight decreases in left renal function demonstrated on renal scan were not picked up on the IVP. All patients who showed a decrease in renal function following left renal vein ligation had a preoperative CrCl less than 100 ml/min. However, this two-hour CrCl was not a split function test, but was taken from a mixed urine specimen from both kidneys. Patients who initially showed diminished or no function of the left kidney all improved and were found to have normal renal function on renal scan within two months postoperatively.

This study confirms that ligation of the left renal vein is well tolerated if necessary for additional exposure during surgery of abdominal aortic aneurysms. Ligation should be carried out close to the inferior vena cava to preserve collateral venous channels. Although a slight decrease in left renal function is demonstrated in a small number of patients, this is not clinically apparent and appears to be insignificant. The usefulness of renal scans in the postoperative period is emphasized in this study since they were found to be the most sensitive indicators of slight changes in renal function. These changes appear to follow the patterns seen with renal vein thrombosis.[8]

If no function is demonstrated on renal scan in the postoperative period when the left renal vein has been ligated, and the patient has no symptoms to suggest renal artery occlusion (such as uncontrollable hypertension, fever, flank pain), additional procedures, such as an arteriogram, may be avoided and the patient followed expectantly. As seen in two patients in our series who had no demonstrable function of the left kidney following left renal vein ligation, renal function returned to normal within two months *(Table II)*.

Renal scans provide additional data by demonstrating segmental or regional perfusion. In one patient (not included in this study) with uncontrollable hypertension postoperatively, patchy perfusion defects were noted in the renal scan. This was felt to be consistent with atherosclerotic emboli from proximal aortic crossclamping, and symptoms resolved during a two week period.

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Cancer Mortality Excess

Evaluation in Counties of Kansas

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JOSEPH G. HOLLOWELL, JR., M.D.,† *Topeka*

Cancer mortality for white residents of Kansas was analyzed for the period 1973-1977, and the results were compared to previously published results for 1950-1969. Cancers significantly high in both time periods include lung cancer in males in Wyandotte and Cherokee counties. Pancreatic cancer is no longer statistically significantly elevated in Barton and Stafford Counties.

RECENT STUDIES by Mason *et al.*[1, 2] determined age adjusted cancer mortality rates for the contiguous United States for the period 1950-1969. Variations in cancer mortality by geographic location were quite striking. Color coded maps were used to identify cancer mortality "hot spots," and to encourage the investigation of specific cancers in counties with excessively high rates. During 1950-1969, cancer mortality rates in Kansas were significantly elevated for a number of sites. For example, pancreatic cancer in white males was elevated in Barton and Stafford counties. Lung cancer mortality rates in white males were elevated for Cherokee and Wyandotte counties. This paper presents the results of recalculating the cancer mortality rates in Kansas for a more recent time period (1973-1977). The principal aim was to assess which cancer sites and counties remained consistently significantly high.

Materials and Methods

Cancer mortality rates were calculated for 16 cancer sites for each of the 105 counties in Kansas, and for 21 cancer sites for each of the 11 county groups. These county groups are the same as the U.S. Bureau of Economic Analysis' State Economic Areas; namely, one county or a group of contiguous counties with similar economic and social characteristics *(Table I)*. The basis for grouping sites according to either individual county or county group was to provide consistency with the 1950-1969 study by Mason *et al.* The same boundaries were used for county groups as in Mason's study.

The data were restricted to white male and female residents of Kansas. Age, sex, and race specific mortality rates were calculated using 18 age categories (0-4, 5-9 . . . 75-79, 80-84, and 85 and over). The population chosen for the denominator of the specific rates was obtained from a 1975 U.S. census bureau estimate made available by the National Cancer Institute. The three digit death coding format for underlying cause of death was based on the International Classification of Diseases Adapted for Use in the United States, Eighth Revision (ICDA-8),[3] after checking for comparability with ICDA-6 used by Mason *et al.*

Age adjusted mortality rates were calculated using the direct method, applying age, sex, race, and site specific county and county group rates to the total 1960 U.S. population (all races, sex specific) (Colton, 1974).[4] Rates were rank ordered for each county or county group for each ICDA code number. If the rate for a county (or county group) minus two standard deviations did not overlap with the state rate plus two standard deviations, a statistically significant ($p < 0.05$) elevated rate was indicated. The method used was generally the same as that of Mason (Chiang, 1961).[5]

For each county and county group, comparisons were made to the 1960 age adjusted U.S. rate using 1973-1977 deaths. Standard deviations for the 1973-1977 U.S. mortality rates are not yet available. Hence, state standard deviations were used as a substitute for U.S. standard deviations when comparison was made to elevated county or county group rates.

Counties with elevated rates in 1950-1969 were compared with the results for 1973-1977 to see if they remained consistently high for the same cancers.

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TABLE I
LISTING OF COUNTY GROUPS, INCLUDING FEDERAL COUNTY IDENTIFICATION CODES, KANSAS

1. Wyandotte (209), Johnson (091)
2. Shawnee (177)
3. Sedgwick (173)
4. Nemaha (131), Brown (013), Atchison (005), Jackson (085), Jefferson (087), Douglas (045), Leavenworth (103), Doniphan (043)
5. Norton (137), Phillips (147), Smith (183), Jewell (089), Republic (157), Washington (201), Marshall (117)
6. Osage (139), Franklin (059), Miami (121), Coffey (031), Anderson (003), Linn (107), Woodson (207), Allen (001), Bourbon (011)
7. Wilson (205), Neosho (133), Crawford (037), Montgomery (125), Labette (099), Cherokee (021)
8. Pottawatomie (149), Riley (161), Geary (061), Wabaunsee (197), Morris (127), Lyon (111), Chase (017), Greenwood (073), Butler (015), Cowley (035), Elk (049), Chautauqua (019)
9. Cloud (029), Clay (027), Ottawa (143), Dickinson (041), Saline (169), Rice (159), McPherson (113), Marion (115), Reno (155), Harvey (079), Kingman (095), Harper (077), Sumner (191)
10. Cheyenne (023), Rawlins (153), Decatur (039), Sherman (181), Thomas (193), Sheridan (179), Graham (065), Rooks (163), Osborne (141), Mitchell (123), Gove (063), Trego (195), Ellis (051), Russell (167), Lincoln (105), Lane (101), Ness (135), Rush (165), Barton (009), Finney (055), Ellsworth (053), Hodgeman (083), Pawnee (145), Edwards (047), Stafford (185), Kiowa (097), Pratt (151)
11. Wallace (199), Logan (109), Greeley (071), Wichita (203), Scott (171), Hamilton (075), Kearny (093), Stanton (187), Grant (067), Haskell (081), Gray (069), Ford (057), Morton (129), Stevens (189), Seward (175), Meade (119), Clark (025), Comanche (033), Barber (007)

Results

The only consistently elevated cancer sites in Kansas were for lung cancer in males in Cherokee and Wyandotte counties. These rates were elevated 54 per cent in Cherokee County and 25 per cent in Wyandotte County over the U.S. average. No cancers were consistently elevated for females. *Table II* shows age adjusted annual death rates for lung cancer in white males in selected counties over time. As shown, the rates for male residents of Cherokee and Wyandotte counties during both time periods were significantly higher than those of both Kansas and the United States. The more recent rates are based on 65 and 258 deaths, respectively. The rate for Crawford County is almost as high as that for Wyandotte County. However, due to fewer deaths it is not statistically significantly elevated. The percentage change in the death rate for males between the two time periods was only slightly greater for Cherokee County than that for the state as a whole and was comparable with those of some of the other counties — particularly the more rural ones. *Table III* shows age adjusted death rates for lung cancer in white females. There were no counties significantly elevated. In Cherokee County, the percentage change between the two time periods was greater than either for the state or for similar sized rural counties. The mortality rate changed from 3.6 to 18.9/100,000/year in Cherokee County. Although this increase is 425 per cent — which is three times greater than the increase of the state was a whole — it should be recognized that the number of female cases is small (12 in 1950-1969 and 15 in 1973-1977), and the death rates are consequently unstable. The sharp increase could, therefore, represent a chance occurrence.

Female breast cancer mortality rates have remained relatively constant throughout Kansas. In 1950-1969 the rate was 23.1/100,000 compared with 24.2/100,000 in 1973-1977. In Cherokee County the breast cancer mortality rates for the same two periods were 19.4 and 19.5/100,000, respectively, practically constant; these rates were among the lowest in Kansas. Thus, the rate for lung cancer was comparable to that for breast cancer (the leading cause of cancer mortality among Cherokee County women) in 1975.

Cherokee County is rural (white population $\simeq$21,000) with an occupational focus today on agricultural production (soybean, corn, wheat, and cattle). However, in the past there was considerable mining of coal, lead and zinc, as well as lead and zinc smelting. In 1970, it ranked about average in population among Kansas' 105 counties. The largest cities in Cherokee County were Baxter Springs (population about 5,100), Galena ($\simeq$3,900), and Columbus ($\simeq$3,650).

Tables II and *III* also list a number of other counties for comparative purposes. Wyandotte County consists largely of Kansas City, Kansas (white population $\simeq$140,000). Johnson County is a suburban county in the Kansas City metropolitan area with a white population of approximately 240,000 in 1970. Sedgwick County contains Wichita, the largest city in Kansas (white population $\simeq$310,000). Shawnee County contains Topeka, and has a white population of about 143,000. Ford County is similar in size to Cherokee County (white population $\simeq$23,000), but is farming oriented and without mines. Franklin, Labette, and Sumner counties are

TABLE II
AGE ADJUSTED[a] LUNG CANCER[b] RATES IN SELECTED KANSAS COUNTIES
FOR WHITE MALES: 1950-1969 and 1973-1977

County of Residence	1950-1969 Number of Deaths	1950-1969 Average Annual Death Rate Per 100,000	1973-1977 Number of Deaths	1973-1977 Average Annual Death Rate Per 100,000	Percent Change in the Average Annual Death Rate Over 15 Years
Cherokee	125	47.1*	65	95.6*	103.0
Crawford	197	38.8	90	75.0	93.3
Wyandotte	610	45.6*	258	78.0*	71.1
Sedgwick	862	38.8	465	69.3	78.6
Johnson	296	31.5	247	58.1	84.4
Shawnee	401	35.1	193	60.3	71.8
Ford	42	20.3	26	47.2	132.5
Franklin	66	26.4	42	69.1	161.7
Labette	106	31.7	37	47.5	49.8
Sumner	95	30.9	52	71.7	132.0
State	6,206	29.5	3,276	57.5	94.9
U.S.	571,226	38.0	285,127	62.3	63.9

[a] Total 1960 U.S. population (all races and sexes combined).
[b] 8th Revision ICDA: Code 162. 6th Revision ICDA: Code 162, 163.
* County rate significantly elevated ($p < 0.05$) above U.S. rate.

also similar in population size to Cherokee County, and are farming oriented. Crawford County, just north of Cherokee County, is an old coal mining area, and has a white population of about 37,000.

Cancers nonsignificantly elevated in Kansas for 1973-1977 which were significantly elevated in 1950-1969 can be found in *Table IV*. These include male pancreatic cancer which is elevated 84 per cent above the U.S. rate in Barton County. This is based on 14 deaths. Stafford County (not shown) is now below the state and U.S. average. This is probably due to the incorrect listing of pancreatic cancer as the

TABLE III
AGE ADJUSTED[a] LUNG CANCER[b] RATES IN SELECTED KANSAS COUNTIES
FOR WHITE FEMALES: 1950-1969 and 1973-1977

County of Residence	1950-1969 Number of Deaths	1950-1969 Average Annual Death Rate Per 100,000	1973-1977 Number of Deaths	1973-1977 Average Annual Death Rate Per 100,000	Percent Change in the Average Annual Death Rate Over 15 Years
Cherokee	12	3.6	15	18.9	425.0
Crawford	26	4.6	17	13.4	191.3
Wyandotte	102	6.5	67	15.8	143.1
Sedgwick	160	5.8	113	12.6	117.2
Johnson	72	6.3	81	14.6	131.7
Shawnee	92	6.3	58	13.4	112.7
Ford	14	6.0	3	4.5	−25.0
Franklin	15	4.9	10	11.9	142.9
Labette	16	4.2	10	9.2	119.0
Sumner	25	7.7	9	8.8	14.3
State	1,205	5.0	823	11.9	138.0
U.S.	108,326	6.3	86,455	15.2	141.3

[a] Total 1960 U.S. population (all races and both sexes combined).
[b] 8th Revision ICDA: Code 162. 6th Revision ICDA: Code 162, 163.

TABLE IV
NON-SIGNIFICANTLY ELEVATED CANCER SITES IN KANSAS

Site	County or County Group (SEA)	1973-77 Ratio of Mortality Rates (Age Adjusted, All Races, Sex Specific) County/U.S.
Male		
Pancreas	Barton	1.84 (14)*
Lip	SEA 9	2.60 (2)
Nasopharynx	SEA 3	1.47 (3)
Thyroid	SEA 3	1.47 (3)
Connective Tissue	SEA 3	1.02 (7)
Leukemia	SEA 10	1.14 (66)
Female		
Cervix Uteri	Cherokee	1.61 (4)
Corpus Uteri	Logan	1.45 (1)
Nose, nasal cavities and sinuses	SEA 7	1.80 (2)
Melanoma of skin	SEA 1	1.07 (15)

* Numbers in parentheses refer to the frequency of deaths during 1973-1977 in each County or County Group.

TABLE V
NEWLY SIGNIFICANTLY ELEVATED CANCER SITES IN KANSAS

Site	County or County Group (SEA)	1973-77 Ratio of Mortality Rates (Age Adjusted, All Races, Sex Specific) County/U.S.
Male		
Total	Wyandotte	1.17 (744)*
	Barton	1.21 (187)
	Marshall	1.24 (129)
Stomach	Jackson	3.40 (10)
Kidney	Butler	2.60 (13)
Bladder	Shawnee	1.72 (39)
Pancreas	Atchison	2.94 (13)
Other Skin	SEA 5	4.73 (10)
Leukemia	SEA 5	1.88 (33)
Lymphosarcoma and Reticulosarcoma	SEA 11	1.84 (29)
Female		NONE

* Numbers in parentheses refer to the frequency of deaths during 1973-1977 in each County or County Group.

cause of death in Stafford County patients during 1950-1969. Upon death certificate review, approximately 50 per cent of deaths attributed to pancreatic cancer could not be verified by hospital or other medical records (Baker *et al.*, 1980).[6] Cherokee County is nonsignificantly elevated 61 per cent above the U.S. rate for cancer of the cervix uteri (4 deaths).

Certain cancer sites have increased in 1973-1977 to become statistically significantly elevated where previously they were not. These can be found in *Table V*. In Butler County, male kidney cancer is now elevated 160 per cent above the U.S. average. This is based on 13 deaths. In Shawnee County, male bladder cancer is now elevated 72 per cent above the U.S. average (39 deaths). Pancreatic cancer is now 194 per cent above the U.S. average in males in Atchison County (13 deaths). No significant excesses are found for females. It should be stressed that a small number of cases will lead to an instability in rates. Some of the excess in *Table V* could be the result of statistical flukes.

Discussion

The only consistently high statistically significant mortality rates found in Kansas during both 1950-1969 and 1973-1977 were for lung cancer in white males in Cherokee and Wyandotte counties. Lung cancer tends to be increased in urban areas nationwide. However, not all lung cancer excess is confined to urban areas. Thus, while it is not unusual to find the excess in Wyandotte County, it is unusual to find the excess in rural Cherokee County. Lung cancer mortality rates are rising more rapidly than expected in females in Cherokee County and lung cancer is now comparable to breast cancer as the leading cause of female cancer mortality. However, the instability of rate changes when dealing with small numbers is recognized. Somewhat similar results have been found for Jasper County, Missouri — across the state line from Cherokee County — in a separate study.

Unlike the 1950-1969 NCI study, this investigation used Code 162 for lung cancer, and did not include Code 163. Code 163 is unspecified as a primary or secondary site in the sixth revision. There is good comparability in death certificate coding for primary site lung cancer between the time periods studied. The population used herein was that estimated for 1975, not the sum of the twenty year's population used by NCI. The principal method of age adjustment used herein was sex specific, although when comparison was made to Mason's data both sexes were combined.

It would be useful to obtain the 1973-1977 cancer rates needed to qualify each site to be in the top 10 per cent nationally. Although such rates have been calculated herein for Kansas, they are not readily

available for the entire U.S. The acquisition of national standard deviations for the period 1973-1977 would also be desirable. The use of Kansas' state standard deviation as a substitute may create false negatives.

The approach used in this study to find statistically significant elevation by site is a conservative one. Cancer sites remaining statistically significant in both time periods ($p < 0.05$ in both 1950-1969 and 1973-1977 equals $p < 0.05$,[2] or $p < 0.0025$, assuming independence) are very unlikely to have occurred by chance and are considered to be of highest priority for further investigation. This method is not the most useful one for identifying excesses of the rarest tumors since deaths during a five year period may yield too few cases in a given county or county group to separate the results from chance occurrence.

One might question the degree to which a cancer mortality rate excess in this study is a reliable indicator of an incidence rate excess. Since the incidence of cancer is a better measure of risk than mortality, particularly for cancers with a low case fatility rate, the use of mortality rates may be questioned. However, Kansas does not have verified cancer incidence data available for the entire population of the state. In a separate study in Iowa — which has a statewide cancer registry — comparisons of mortality with incidence rates revealed some congruence for lung cancer. We believe that with cautious interpretation, the lung cancer mortality rate can be used to indicate risk in Kansas.

Possible risk factors for lung cancer among whites (other than increasing age) include (Fraumeni):[7] cigarette smoking, pulmonary fibrosis (*e.g.*, scarring from tuberculosis), arsenic, asbestos, cadmium, and uranium. Lung cancer is the most common tumor related to an airborne environmental determinant. Because of the continued significantly elevated mortality rates for lung cancer in Cherokee and Wyandotte Counties, we are undertaking additional studies to verify death certificates and to ascertain whether risk factors unique in Cherokee and Wyandotte Counties may be contributing to the increased lung cancer mortality rate.

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Acknowledgement

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Immune Deficiency States — Evaluation and Management

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ALTHOUGH PRIMARY immune deficiency states are rare, the practicing physician may encounter several cases of secondary or acquired immune deficiency. Briefly reviewed here are practical aspects in the diagnosis and therapy of immune deficiency states in humans. Comprehensive reviews and case illustrations have been published.[1-8]

Host defense abnormalities could be due to defects in any of the following systems: (1) Thymus dependent (T) system — responsible for cell mediated immunity and defenses against viral, fungal, and parasitic infections; (2) Bone marrow dependent (B) system — responsible for immunoglobulin-antibody synthesis and for defenses against bacterial infections; (3) Combined T and B systems; (4) Complement system; (5) Phagocytic system; or (6) Secretory immunoglobulin-mucosal system.

The pathogenesis of immune deficiencies can be (1) congenital due to maturation development defects in any of the above systems, or (2) acquired and secondary to administration of drugs (corticosteroids, cytotoxic agents), advanced malignancy — *e.g.* multiple myeloma; granulomatous diseases — *e.g.* sarcoidosis; protein-losing states — *e.g.* malabsorption, burns, nephrotic syndrome; systemic metabolic abnormality — *e.g.* diabetes mellitus, uremia, alcoholism; or post splenectomy or aging.

Several associations can be seen in patients with immune deficiency states. These include autoimmune diseases, allergies and various malignancies, particularly those of the lymphoid system and the gastrointestinal tract.

Evaluation

Clues to the presence of a possible immune deficiency state include: recurrent infections, particularly if they are sinopulmonary, ear, or skin infections; incomplete resolution of infections following therapy or rapid recurrence of infections following the discontinuation of antibiotic therapy; infections with opportunistic or unusual infectious agents; pulmonary infection in nonsmokers in the absence of apparent cause; and association of infections with diarrhea, allergy, or collagen vascular disease.

Table I lists tests for diagnosis of various immune deficiency states. These are divided into those that can be done in a physician's office or small community hospital and those that usually are done in a large referral center. Simple tests are extremely valuable and give a clue to or confirm the diagnosis in many patients.

Clinical Features

There are predominant features for each type of immune deficiency. Overlap of clinical features and involvement of more than one system are frequently seen. A summary of those features is shown in *Table II*.

Therapy

Therapy is dependent on the type of immune deficiency, the predominant infection, and the severity of the infection at the time of diagnosis. The following general guidelines are helpful.

• *Aggressive and early treatment of infections.* Antibiotics could be used continuously, intermit-

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TABLE I
EVALUATION OF IMMUNE DEFICIENCY

System	*Simple Tests (Normal values)*	*Elaborate (Research) Tests*
B	Isohemagglutinins (Titer >1:8, in O, A, B blood group patients)	B cell numbers (Surface Ig assay)
	Serum quantitative immunoglobulin assay (Consult reference values for children. Adult values are IgG 650-1500 mg/dl, IgA 70-325 mg/dl, IgM 60-350 mg/dl)	B cell immunoglobulin biosynthesis
	Bone marrow examination for plasma cells (present)	Secondary humoral response (tetanus, diphtheria)
T	Delayed skin test (PPD, mumps, monilia) (at least one is positive = induration >8 mm at 48 hrs)	T cell numbers (E rosette)
	Blood lymphocyte count	In vitro T cell mitogen-induced blastogenesis
		Production of lymphokines
		T cell subpopulations by monoclonal antibodies
Complement	CH_{50}, C_3, C_4 assay (CH_{50} 130-230 units; C_3 85-180 mg/dl; C_4 15-45 mg/dl)	Functional assay of complement components and their inactivators
		Quantitation of various complement components
Phagocytic	Granulocyte and monocyte blood counts	Phagocytic index
		Nitro blue tetrazolium test
		Chemotaxis and random migration
		Bactericidal assay
		Tests for intracellular metabolic events

tently, cyclicly, or only when needed. The decision about which antibiotic, how often, and how long should be tailored to each patient. Broad spectrum bactericidal antibiotics are valuable.

- *Supportive therapy*. Proper drainage of lung, ear, or sinuses may avoid permanent damage to these organs.
- *Immunization*. Live virus vaccines are absolutely contraindicated; however, pneumococcal and influenza vaccines are valuable.
- *Reconstitution therapy*. Reconstitution of the immune system can be curative in certain patients, supportive in others, and harmful in some. Proper knowledge of the system involved and the probable risks of therapy are important.

In adult hypogammaglobulinemic states — particularly those with serum IgG levels less than 200 mg/dl — human pooled immune serum globulin (ISG) is indicated. A dose of 0.5 mg/kg/3 wks given intramuscularly is the routine dose. A loading dose

TABLE II
CLINICAL FEATURES OF VARIOUS IMMUNE DEFICIENCY STATES

System Involved	*Predominant Clinical Features*
B	1. Recurrent bacterial infections (lung, sinus, ear, skin) 2. Diarrhea, malabsorption, nodular lymphoid hyperplasia of gut 3. Common associations: allergies, autoimmune syndromes
T	1. Viral and fungal infections 2. Common associations: granulomatous diseases
Phagocytic	1. Recurrent bacterial or fungal infections 2. Common associations: granulomatous disease
Complement	1. Recurrent bacterial infections. Neisseria infections in C6-9 deficiency 2. Common associations: collagen vascular disease
Secretory Ig	1. Mucosal ulcers 2. Intestinal candidiasis

twice the routine dose should be given at the initiation of therapy and when the patient acquires a serious infection that is not responding to proper antibiotic therapy. Adequate ISG therapy should increase the patient's serum IgG level to greater than 250 mg/dl. Systemic anaphylactic or anaphylactoid reactions occur in a few patients. These reactions are usually due to aggregates present in the ISG or in the patient who develops antibodies against the administered ISG. ISG clearly is contraindicated in patients with isolated IgA deficiency since the incidence of anaphylactic reactions upon ISG administration is high in these patients. ISG is of no proven value in patients who have recurrent infections but with normal serum IgG levels. Moreover, in infants with transient delayed hypogammaglobulinemia, who start to synthesize their own immunoglobulins at age 2-3 years instead of 6 months, administration of ISG is not advisable because exogenous ISG might turn off the synthesis of immunoglobulins by these patients. Intravenous ISG is commercially available (Gamimune, Cutter Laboratories). ISG contains IgG only; therefore patients whose immune deficiency state requires reconstitution with IgM, IgA or complement components, should be given intravenously fresh frozen plasma from appropriate donors. The latter should be matched, free of hepatitis antigen or other infectious agents that could be transmitted and, preferably, selectively immunized to provide the specific antibody to the recipient. The dose of plasma is 10 ml/kg/3 wks, and the dose or frequency could be increased if needed. The goal of therapy is to achieve a serum IgG level of 250 mg/dl.

In patients whose immune deficiency is due to thymic gland hypoplasia — as in DiGeorge syndrome — fetal thymic transplants or thymic epithelium transplant can be curative. Trials of administration of thymic hormones to these patients are in progress.

Transfer factor is a dialysate prepared from leukocytes of normal sensitized individuals. It probably is of questionable value in immune deficiency states except for its beneficial value in some cases of chronic mucocutaneous candidiasis when given in conjunction with antifungal drugs.

Bone marrow transplantation from a matched donor is the proper therapy for severe combined B and T cell immune deficiency states. Results of fetal liver grafts in these patients have been disappointing. Administration of matched normal red cell transfusion to a subgroup of these patients with adenosine deaminase deficiency has been valuable.

In conclusion, practicing physicians should familiarize themselves with the fundamental knowledge of immune deficiency states, how to diagnose the defect and the system involved, and the proper care of these patients. The impact of modern immunology on this group of diseases is enormous, and the future is bright for these patients.

Self-Assessment Questions

For each question, more than one answer may be true.

1. In primary hypogammaglobulinemia, the following tests are helpful for evaluating B cells:
 a. Nitroblue tetrazolium test
 b. Isohemagglutinin titer
 c. In vitro phytohemagglutinin response of blood lymphocytes
 d. Tetanus antibody titer following booster immunization
2. The following infections are usually seen with T cell deficiency states:
 a. Viral
 b. Bacterial
 c. Fungal
 d. Parasitic
3. Selective IgA deficiency could be seen in or in association with:
 a. Healthy individuals
 b. Allergy states
 c. Sinopulmonary infections
 d. Granulomatous diseases
4. Statement about treatment of immunodeficiency:
 a. Bone marrow transplantation is curative for B cell deficiency.
 b. Thymus transplantation is curative for DiGeorge syndrome.
 c. Gamma globulin is contraindicated in IgA deficiency.

(Answers on page 260)



Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Beta-Adrenergic Blockers

Question: Would you provide me with information regarding the use of beta-adrenergic blockers to prevent myocardial reinfarction?

Answer: Beta-adrenergic blockers have been used successfully for many years to treat a variety of cardiovascular and non-cardiovascular disorders. Their use in the prevention of myocardial reinfarction has been a subject of much controversy. Recently, three major studies have been reported which clearly indicate improved survival rates in post myocardial infarction patients receiving beta-adrenergic blockers. The results of these studies are detailed below.

Timolol study. A Norwegian study using timolol (Blocadren) demonstrated a substantial reduction in mortality and reinfarction in patients surviving a myocardial infarction. Compared to placebo, timolol started seven to 28 days after infarction reduced the mortality rate by 45 per cent. The drug was continued for up to 33 months. Timolol was administered in an oral dose of 10 mg twice daily.[1]

Propranolol study. A large, multicenter trial in the United States conducted by the National Heart, Lung, and Blood Institute was stopped nine months early because patients receiving propranolol (Inderal) exhibited a 26 per cent lower mortality rate than did patients receiving placebo following myocardial infarction. The drug was started five to 21 days post infarction and continued for up to 30 months. The continuation of the trial was deemed unethical because of the obvious difference between the survival rates of the propranolol group and the placebo group. Propranolol was administered in an initial oral dose of 40 mg three times daily and was adjusted to either 60 mg or 80 mg three times daily after one month, based on serum propranolol levels.[2]

Metoprolol study. Metoprolol (Lopressor) was studied in a Swedish trial, beginning immediately following patient admission to the hospital and continuing for a period of 90 days. In comparison with placebo, a 36 per cent reduction in mortality rate was noted with metoprolol. Following an initial intravenous dose, a 100 mg dose of metoprolol was administered orally twice daily.[3]

These agents have demonstrated improved survival rates following myocardial infarction. Further research will be required to better define optimal dosage regimens and to identify which patients are most likely to benefit from beta-adrenergic blocker therapy. Timolol is presently the only agent in this group with an FDA-approved indication for use in myocardial reinfarction prophylaxis. Propranolol, metoprolol, and perhaps other beta-adrenergic blockers will surely achieve this indication in the near future. *Table I* illustrates the results of the three major trials.

Submitted by
Steven B. Cano, Pharmacy Resident

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

TABLE I

	Patients	*Mortality*	*Benefit*
Timolol study			
Placebo	939	13.9%	
Drug	945	7.7%	45%
Propranolol study			
Placebo	1921	9.5%	
Drug	1916	7.0%	26%
Metoprolol study			
Placebo	697	8.9%	
Drug	698	5.7%	36%



Editorial COMMENT

Trouble from Paradise

We admit to a strong sense of tradition but, at the same time, a realization that it may carry us down unintended roads at times. The example of the moment is the Eden story with its generally accepted message that sex caused our first and most profound fall from grace, entitling that function to the dubious honor of being classified as Original Sin. In our reading of the matter, however, the truly *original* sin preceded and begat the more lurid event.

Consider. Here was the ideal existence — congenial surroundings, food — whatever was necessary, shelter — as necessary, clothing — not necessary — in short, the ultimate in social security. We look back on this state of total comfort and call it Paradise, envying the man's freedom from having to do anything, even think — *especially* think. There is no evidence that he was dissatisfied (he hadn't thought about it) but this was decided for him with the result that he was provided in one maneuver with thoracic symmetry and company for dinner.

Considering his innocence and inexperience, he must have noticed that this interloper embodied many similarities to himself but, at the same time, some disturbing differences. It didn't require carnal implementation to produce the upheaval. Rather, it set him to thinking and *that* was the original sin (complete with the female there to blame it on). To this day, the corrupting process plagues their children — not just the process but its most distressing product, the realization that the spirit and the substance, once comfortably singular, are now distinct entities eternally in conflict with each other. Paradise, it seems, was the total harmony of these two elements, a state of common identity which rendered it unnecessary to give any thought as to which took precedence.

If this opening gambit comes across as frivolous, it introduces a subject that is assuredly serious — abortion — which, as much as any of mankind's problems, epitomizes the continuing challenge to resolve the spiritual and material stresses. It has been suggested in the past that we address the subject but we have been reluctant to do so for two reasons. First, the matter has inspired reactions of such intensity and volume as to make it unlikely that anything new can be added — or opinions changed. Attitudes have become so polarized that one needs to hear only a few words before one knows the speaker's intent — either the emotional charges of murder and butchery or the impassioned call for uninhibited license. But our personal problem is that we are unable to identify fully with either extreme and are consequently condemned to the limbo of moderation, denied the one pole or the other where the sin of thought has been neutralized by the absolution of righteous certainty.

We admit that our failure of reconciliation with the dogma of either extreme betrays a sad lack of character but it is our contribution to maintaining this world in its imperfect state — else, as has been said, what's a heaven for? In the first place, the equation of the rights of the conceptus with those of the mother is, for us, patently impossible. To deny that any justification for abortion exists is to place its interests above those of the mother (without whom it would not exist). Furthermore, this interpretation excludes the medical intellect from any role or purpose in a matter of considerable medical concern.

We submit to no one in our admiration for the reproductive process and consider it one of the prime factors in the development of mankind's theistic philosophies since the implication of immortality is interpreted by its human agents as a link with divinity. We bring forth children not only in sorrow but in imitation. But the mergence of the pronuclei, governed as it is by the laws of chemical affinity (God-given laws, if you will), is a step in the process, albeit an important one. To identify this tentative arrangement (in which the promise and the threat come as near equivalency as anything in the biological world) as the accomplished fact of human worth and personality is to negate all subsequent natural and human (again God-given) endeavor. The conceptus at this point is on a par with many such

processes in the animal world, none of which, in a fine exercise of self-preservation or conceit, mankind extends anything like such consideration except when commercial factors are involved or in a belated attack of conscience as when a species faces extinction.

So that puts us solidly on the side of free-for-all abortion? Well, no — that might be a comfortable resolution of our condition but extremes, although using differing frameworks and terminology, have the same generic base. For one thing, the feminist insistence that the procurement of abortion on demand is an expression of a woman's right to exercise control over her own body ignores the rights of those professional personalities who may be called upon to implement the action. The do-it-yourself procedures have a long and highly unsatisfactory history and it is impossible to consider the rights of one without considering the rights of all involved. The assessment of abortion as a sin lies in its power to corrupt when it fails to use the highest exercise of human intellect — really our only defense in this unsettled world we have inherited. Paradoxically, the social and domestic upheaval that human reproduction can create can be a mechanism for the destruction of human values. The elimination of the illegitimate abortionist by legal fiat (although never complete) has made for legal but still illegitimate performers who function primarily for financial gain and are, despite professional qualification, as reprehensible. The case histories of physical and emotional trauma, however, are not a new acquisition. They have been present but unrecorded since the clandestine activities of the past, while less frequent, were more devastating.

It is apparent that inherent in the performance of abortion is the obligation to arrive at that performance only after the full application of medical service — the determination of the fullest justification, the preparation and support of the patient, professional conduct of the service, and continued guidance and support afterward. The extent to which these requirements are fulfilled measures the medical legitimacy of the procedure as fully as legal sanction, and the degree to which they are not fulfilled lends credence to the critics — and plenty of ammunition.

So it is that our personal resolution of the conflict (and let it be noted that we speak throughout these remarks not for any other person or any organization) would be viewed by many as simplistic and evasive. Our acknowledgement that abortion can be justified automatically condemns us from the one side (which, not incidentally, includes many physicians) and our belief that such justification is not a mandate for unrestrained use of abortion alienates us from the other extreme. But our stand, for better or worse, is and has been the belief that abortion is a form of medical therapy to be arrived at in the traditional manner of any medical problem: the presentation by the patient of a medical condition, its evaluation and interpretation by a physician on medical merits, and management by justifiable medical methods with full understanding by physician and patient as to intent, propriety, and prognosis (insofar as can be determined at the human level since it is the only one mankind can speak for). The term "therapeutic" is all but dropped from our lexicon which is unfortunate since it should be retained as a reminder that the procedure should be conducted in the full sense of medical responsibility. Since the right-to-life people have long since given us up as a lost soul, we can expect to hear from the other side, "How can you ignore the physician's social responsibility?" In our opinion, we have not. What we have done is remove it from the social scientists and return it to the area of medical science where, if physicians function as they should, it is possible to handle such issues without abdication to others.

Our present detachment from the actual scene may be blamed by some for this naive and impractical approach but we are not without our battle scars. It seems a good many years ago, when statutory prohibition of abortion was considered to reflect the true public interest and the word could hardly be uttered in polite society because of its criminal context, that we, along with a number of our colleagues, appeared before a legislative committee to attempt to ease that prohibition. We thought then (and still do) that our purpose was good and our approach moderate. Abortion, a crime, could be justified legally in the extremity of saving a mother's life so the physician was in the position of committing a crime first and hoping to justify it (on medical grounds but against social and legal pressures) after the fact. Our more extensive objection, however, was that, by its prohibitory function, it dictated by statute a form of medical management. This intervention into medical practice, although negative in this particular context, was ominous in precedent since the principle could be applied to positive requirement — including the enforcement of a given morality. We sought not to force anyone to do anything nor to deny them the freedom to choose but to put the decision making in this — and any — basically medical process upon the physician and patient. So we were not a little surprised and nonplussed (naively, in retrospect) to

discover within a day or two we had made the front page of at least one religious newspaper (unfavorably, in case there is any doubt), that friends in this and other communities were contacted by incensed co-religionists wanting more information about this character and, inevitably, that some of our patients wanted no more to do with us.

An era of social tumult separates us from those days and the abortion issue has been at the forefront with increasing rancor on both sides. The reaction we encountered at that earlier time (and many others have since) is reflected in the action of the Attorney General's office which led to the confirmation by the Kansas Supreme Court that the names and fees of those providing medical service to individuals whose care was paid for by the government were public information and available to anyone on request. Since the law has been in effect for some years and applies to all medical services, the interesting feature was that the action was brought by the right-to-life people and directed at the performance of abortions.

To what end? Is a moral hit list to be prepared? Are physicians to be made aware that their actions will be publicized for the adverse effect on them? At the least, it exposes an interesting interpretation of morality.

The first man and woman, later on, must have lived in a particular state of anguish since they had enjoyed, however briefly, the pleasures of Paradise, something their progeny have been able to experience only in dreams. But the anguish for the progeny is not so much in the loss of those comforts or because of the burdens of work thrust upon them. Rather it is in those painful convulsions of the thinking process, translated into the concept called morality, as they have learned that the real punishment is in the irreconcilable conflicts between spirit and substance. — *D.E.G.*

Editorial Note

It has come to our attention that the *Journal of the Kansas Medical Society* recently carried an article that virtually duplicated one appearing in *Missouri Medicine,* the journal of the Missouri State Medical Association, a month earlier. The timing of publication indicates that the paper was under consideration by the two journals at the same time and that prior to publication neither journal was informed of the other's interest in it. Although an element of chance entered into its first publication by *Missouri Medicine,* and that journal has not requested retribution for this breach of editorial principle, we offer our voluntary apology to them and, as well, to our readers who are assured that the articles published in this journal are, to the best of our determination, original and valid without prior publication unless attribution is made otherwise. At the same time, authors are reminded of the unequivocal condition in our "Information for Authors" that papers are to be submitted for our consideration alone, a principle accepted by all reputable journals and providing protection for authors as well as the journals.

Obituaries

Clarence A. Gilmore, M.D., 50, Kansas City, Meharry Medical College 1960, died January 1, 1982.

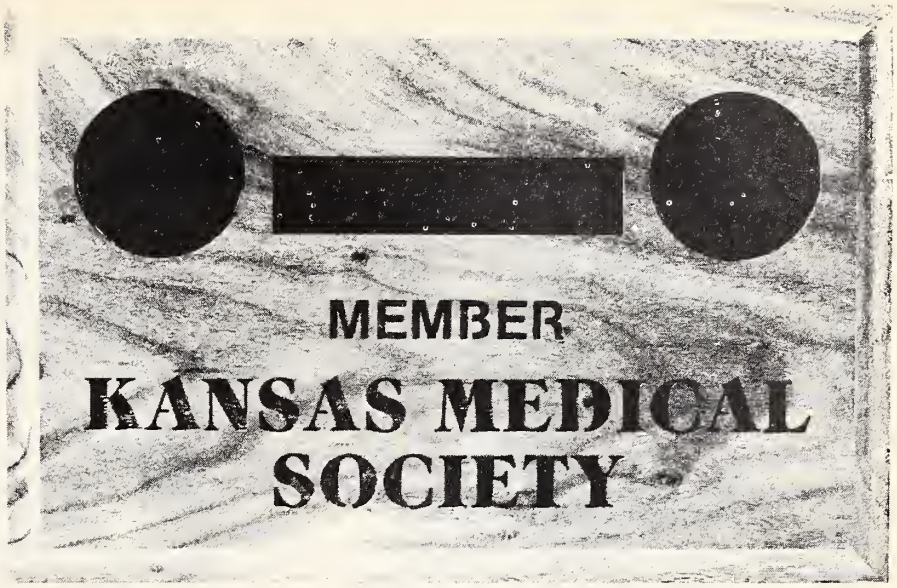
Daniel S. Klassen, M.D., 68, Newton, University of Kansas School of Medicine, died December 10, 1981.

George C. Meek, M.D., 76, Arkansas City, University of Kansas School of Medicine, died March 13, 1982.

Theodore W. Oltman, M.D., 81, Riley, Rush Medical College 1929, died March 16, 1982.

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Tissue Necrosis

(Continued from page 236)

sequence of events and findings at the time of the debridement procedure. This occurred in a setting of profound vascular collapse, which persisted for several hours following the injection. This condition may have increased the likelihood of tissue necrosis, due to diminished local tissue perfusion and oxygen saturation. Although this patient made a complete recovery from his presenting life-threatening acute illness, his course was complicated by severe tissue necrosis of the arm which added substantially to his morbidity and resulted in diminished function of the arm 18 months post-injury.

This case confirms that subcutaneous extravasation of methylene blue may have serious consequences, and should be avoided if at all possible. If methylene blue is to be administered intravenously, particular care care should be taken to insure that the intravenous device is properly placed and freely running, and that blood return can be obtained periodically during the injection.

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Immune Deficiency States

(Continued from page 251)

Answers

1. a, *False;* b, *True;* c, *False;* d, *True;*
2. a, *True;* b, *False;* c, *True;* d, *False;*
3. a, *True;* b, *True;* c, *True;* d, *False;*
4. a, *False;* b, *True;* c, *True;*

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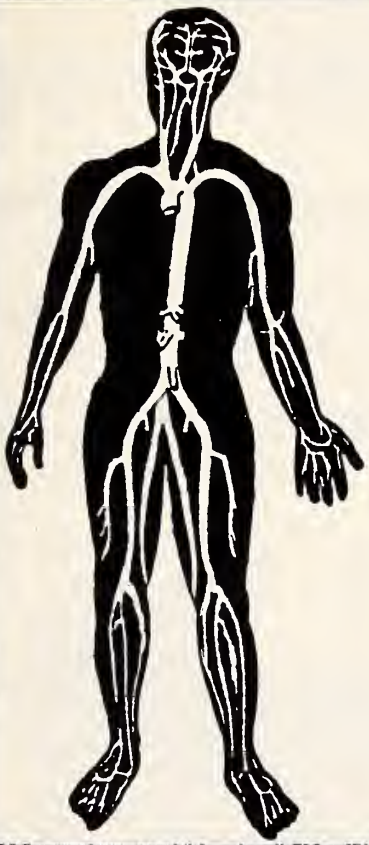
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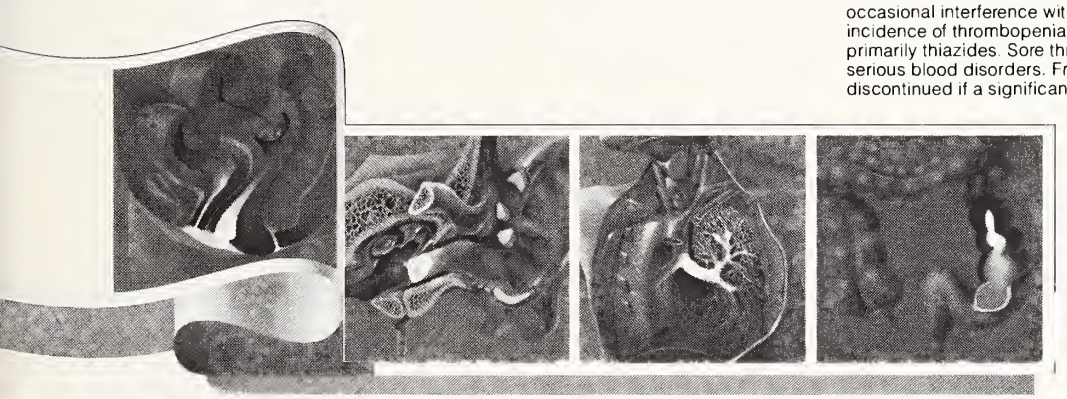
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Join KaMPAC

Bactrim™
(trimethoprim and sulfamethoxazole)
succeeds

Bactrim is useful for the following infections when due to susceptible strains of indicated organisms (see indications section in summary of product information):

Expanding its usefulness in antimicrobial therapy



in recurrent UTI...
a continuing record of high clinical effectiveness against common uropathogens

in acute otitis media in children...
effective against both major otic pathogens...with *b.i.d.* convenience

in acute exacerbations of chronic bronchitis in adults...
clears the sputum and lowers its volume...on *b.i.d.* dosage

in shigellosis...
faster relief of diarrhea than with ampicillin[2]

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. Limited clinical information presently available on effectiveness of treatment of otitis media with Bactrim when infection is due to ampicillin-resistant *Haemophilus influenzae.* To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

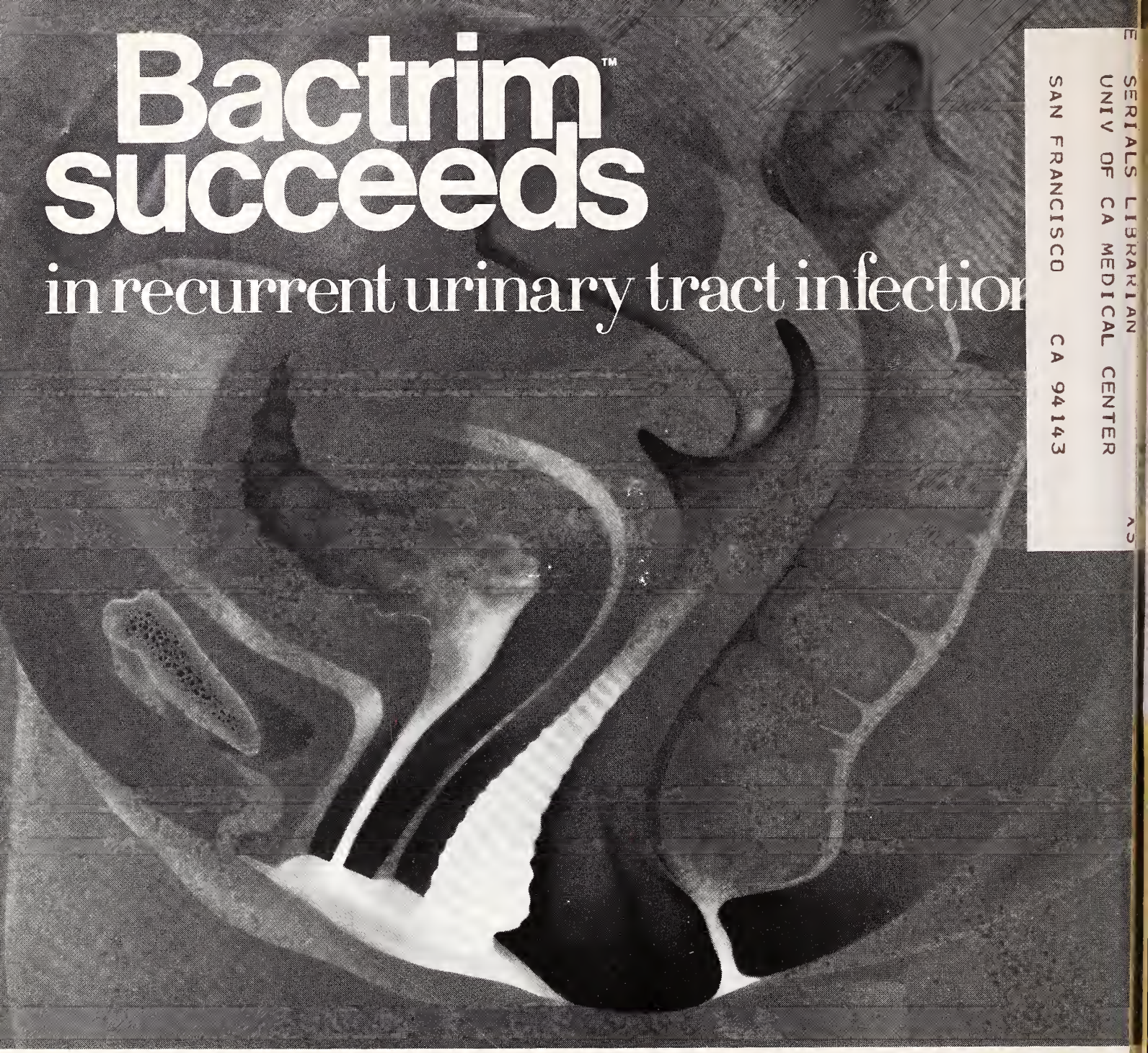
Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry-flavored—bottles of 100 ml and 16 oz (1 pint) *Suspension,* containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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ROCHE LABORATORIES
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110



Bactrim™ succeeds

in recurrent urinary tract infection

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Bactrim continues to demonstrate high clinical effectiveness in recurrent urinary tract infections. Bactrim reaches effective levels in urine, serum, and renal tissue[1]...the trimethoprim component diffuses into vaginal secretions in bactericidal concentrations[1]... and in the fecal flora, Bactrim effectively suppresses Enterobacteriaceae[1,2] with little resulting emergence of resistant organisms.

1. Rubin RH, Swartz MN: *N Engl J Med 303*:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

Bactrim™ DS

160 mg trimethoprim and 800 mg sulfamethoxazole

DOUBLE STRENGTH TABLETS

maximizes results with B.I.D. convenience

ROCHE®

*due to susceptible strains of indicated organisms

Please see previous page for summary of product information.

RESOLUTIONS

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THE Journal OF THE Kansas Medical Society

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1982

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1982 ROSTER

The new roster will be published in August

DO WE HAVE YOUR CORRECT ADDRESS AND PHONE NUMBER?

Please advise us of corrections by June 25.

Send information to:

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Transfer of Power

Herman W. Hiesterman, M.D., Quinter, outgoing president, presents the gavel of office to Kermit G. Wedel, M.D., Minneapolis, who will carry the responsibilities of the presidency of the Kansas Medical Society during the coming year.



Council Meeting

Report of Meeting Held January 17, 1982

A meeting of the Council was held on Sunday, January 17, 1982, at the Salina Hilton Inn, beginning at 10 AM. Herman W. Hiesterman, M.D., President, presided. Present were: Drs. Kenneth Boese, James G. Bridgens, Dean T. Collins, Clair C. Conard, Jack R. Cooper, Louis M. Culp, Herbert D. Doubek, Herbert Fransen, Jimmie A. Gleason, Phillip A. Godwin, Donald D. Goering, Jerry D. Hall, Robert Hughes, Warren E. Meyer, John C. Mitchell, Robert F. Moore, John R. Neuenschwander, Sr., James H. Ransom, Ivan E. Rhodes, Alex Scott, Marvin Snowbarger, Millard C. Spencer, Thomas F. Taylor, Max E. Teare, William K. Walker, Wallace N. Weber, and Kermit G. Wedel. Also present was Mr. Terry J. Nugent, Membership Account Executive-AMA. Also present was Betty Moore (Robert F.), KMS Auxiliary President. Also present were Jerry Slaughter, Gary Caruthers, and Val Braun.

The Council heard a report from the Kansas Council on Economic Education, which has developed a program for educating elementary and secondary level school teachers concerning the basics of the American economic system. The program incorporates economics into classroom teaching by conducting 22-25 classes annually involving approximately 700 teachers, offering resource materials, and covering one-half of tuition for the teachers. The Council approved a $10,000 contribution to the KCEE.

A report on cognitive medicine reimbursement was presented by representatives of the Kansas Society of Internal Medicine. KSIM asked that an ad hoc committee be appointed to develop detailed descriptions of cognitive services and to work with the insurance industry in recognizing and utilizing CPT-4.

It was announced that the recent opinion by the KMS legal counsel on the current status of the Relative Value Study reaffirms the original opinion, and recommends that no further action be taken with respect to the utilization of a relative value scale until the U.S. Supreme Court concludes its study on this issue.

The Council voted to contribute $3,000 to the KaMPAC Education Fund.

The Council heard a report on the current status of six lawsuits in which the Kansas Medical Society is involved. One lawsuit relates to the Department of Social and Rehabilitation Services and the Attorney General, concerning the release of abortion records from the Medicaid program. The Kansas Medical Society filed an *amicus curiae* brief with the SRS. Another suit concerns the Board of Nursing and the Advanced Registered Nurse Practitioner Regulations.

Donald D. Goering, M.D., Chairman of the Membership Recruitment Committee, reported that the Committee will be striving to accelerate the recruitment of student members and new physicians in practice. A peer-to-peer recruitment effort is anticipated.

The Nominating Committee, under the chair-

manship of Phillip A. Godwin, M.D., presented the following slate of nominees:

PRESIDENT ELECT: Jimmie A. Gleason, M.D., Topeka

FIRST VICE PRESIDENT: F. Calvin Bigler, M.D., Garden City

SECOND VICE PRESIDENT: Clair C. Conard, M.D., Dodge City; Lew W. Purinton, M.D., Wichita

CONSTITUTIONAL SECRETARY: K. William Bruner, Jr., M.D., Wichita; Virginia T. Gruendel, M.D., Kansas City

TREASURER: Richard Meidinger, M.D., Topeka; William K. Walker, M.D., Sedan

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Franklin G. Bichlmeier, M.D., Kansas City

AMA DELEGATE: Donald D. Goering, M.D., Coldwater; Alex Scott, M.D., Junction City

AMA ALTERNATE DELEGATE: Warren E. Meyer, M.D., Wichita; Linda D. Warren, M.D., Hanover

The Council heard a report on the status of bills currently in the Legislature.

The Council was advised of the change in the AMA policy with respect to interest charges. The Judicial Council now recommends that it is proper and ethical for physicians to charge interest on overdue accounts if certain guidelines are adhered to. The Council adopted the new AMA policy.

The Council reviewed a complaint about the practice of radiologists reading x-ray films and confirming physicians' findings after the patient has been treated and discharged from the hospital. The Council heard a report from a radiologist with respect to the appropriateness of such physician service. It was determined that misunderstandings of this type are common but that they can be alleviated through concerted patient education efforts on the part of radiologists.

The Council learned that MEDISERVE, Inc. has not issued new student loans for the last two entering classes. At the same time, the state scholarship program has been cut back to a maximum of 100 students per entering class. The Kansas Medical Society advanced a loan of $12,000 to MEDISERVE in 1981. It is anticipated that the revolving loan fund for prior student assistance will reach a self-sustaining level in approximately seven years. The Executive Committee was directed to make a recommendation relative to this program at the next meeting of the Council.

The Council approved the purchase of 200 copies of the book, "Beyond Survival," which deals with stress, substance abuse, and suicide as they relate to physicians. The Impaired Physician Committee will distribute the book to medical students and residents at the University of Kansas School of Medicine-Kansas City. The book will be accompanied by a letter from the KMS President.

The Council approved the expenditure of up to $2,000 for the support of the hospitality suite at the AMA Annual Meeting in Chicago for the purpose of promoting the candidacy of William J. Reals, M.D., for re-election to the AMA Council on Medical Education. These expenditures will come from the President's Fund.

The meeting adjourned at 12:20 PM.

Report of Meeting Held May 2, 1982

The Council of the Kansas Medical Society met on Sunday, May 2, 1982, at the Hilton Inn East, Wichita, subsequent to the Annual Meeting. Those present at the meeting included the following: Drs. Philip J. Antrim, Franklin G. Bichlmeier, F. Calvin Bigler, Kenneth M. Boese, K. William Bruner, Jr., Clair C. Conard, Louis M. Culp, Herbert Fransen, Robert J. Haskins, Robert W. Hughes, James J. Muehlberger, John R. Neuenschwander, Lew W. Purinton, James H. Ransom, Clifton C. Schopf, Alex Scott, G. Rex Stone, Max E. Teare, William K. Walker, Wallace N. Weber, and Emerson Yoder. Also present were Robert D. Durst, Jr., M.D., President of the Kansas Dermatology Society; and Dean T. Collins, M.D., representing the Kansas Psychiatric Society. Also present were Steve Carter, Jerry Slaughter, Gary Caruthers, and Val Braun.

The newly installed President, Kermit G. Wedel, M.D., Minneapolis, presided. He introduced the new Councilors and members of the Executive Committee.

Dr. Wedel announced that the Kansas Medical Society would be contacting those specialty societies that had been inactive during the past year, and encourage them to elect officers. Initially, they

(Continued on page 306)

HYPERTENSION:



METHYLDOPA? RESERPINE? INDERAL? COUNTLESS THOUSANDS WOULD BE BETTER OFF WITH INDERAL® (PROPRANOLOL HCl) B.I.D.

The sooner, the better.

Today, INDERAL—instead of methyldopa, instead of reserpine.

INDERAL exhibits few of the disturbing side effects of methyldopa and reserpine. Sedation, depression, and impotence are rare.* Tolerance is not likely to occur, as it frequently does with methyldopa. For the vast majority of patients—INDERAL means a step toward improving the quality of life. (INDERAL should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.)*

INDERAL blocks beta-receptor sites *in the heart* to reduce heart rate and cardiac output—reducing cardiac work load—sparing an overburdened heart.

Hypertensive hearts can rest easy with INDERAL. For many—it is ideal, first-step therapy.

INDERAL—the sooner, the better for hypertension—a leading risk factor in coronary heart disease.[1]

*Please see following page for Brief Summary of Prescribing Information.



THE MOST WIDELY PRESCRIBED BETA BLOCKER IN THE WORLD

INDERAL®
(PROPRANOLOL HCl)
B.I.D. FOR HYPERTENSION

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

Inderal® BRAND OF **propranolol hydrochloride A beta-adrenergic blocking agent**

BEFORE USING INDERAL (PROPRANOLOL HYDROCHLORIDE), THE PHYSICIAN SHOULD BE THOROUGHLY FAMILIAR WITH THE BASIC CONCEPT OF ADRENERGIC RECEPTORS (ALPHA AND BETA), AND THE PHARMACOLOGY OF THIS DRUG.

CONTRAINDICATIONS

INDERAL is contraindicated in: 1) bronchial asthma; 2) allergic rhinitis during the pollen season; 3) sinus bradycardia and greater than first degree block; 4) cardiogenic shock; 5) right ventricular failure secondary to pulmonary hypertension; 6) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL; 7) in patients on adrenergic-augmenting psychotropic drugs (including MAO inhibitors), and during the two week withdrawal period from such drugs.

WARNINGS

CARDIAC FAILURE. Sympathetic stimulation is a vital component supporting circulatory function in congestive heart failure, and inhibition with beta-blockade always carries the potential hazard of further depressing myocardial contractility and precipitating cardiac failure. INDERAL acts selectively without abolishing the inotropic action of digitalis on the heart muscle (*i.e.*, that of supporting the strength of myocardial contractions). In patients already receiving digitalis, the positive inotropic action of digitalis may be reduced by INDERAL's negative inotropic effect. The effects of INDERAL and digitalis are additive in depressing AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, continued depression of the myocardium over a period of time can, in some cases, lead to cardiac failure. In rare instances, this has been observed during INDERAL therapy. Therefore, at the first sign or symptom of impending cardiac failure, patients should be fully digitalized and/or given a diuretic, and the response observed closely: a) if cardiac failure continues, despite adequate digitalization and diuretic therapy, INDERAL therapy should be immediately withdrawn. b) if tachyarrhythmia is being controlled, patients should be maintained on combined therapy and the patient closely followed until threat of cardiac failure is over.

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuation of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when INDERAL is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease, who are given propranolol for other indications.

IN PATIENTS WITH THYROTOXICOSIS, possible deleterious effects from long term use have not been adequately appraised. Special consideration should be given to propranolol's potential for aggravating congestive heart failure. Propranolol may mask the clinical signs of developing or continuing hyperthyroidism or complications and give a false impression of improvement. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. This is another reason for withdrawing propranolol slowly. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

IN PATIENTS DURING ANESTHESIA with agents that require catecholamine release for maintenance of adequate cardiac function, beta blockade will impair the desired inotropic effect. Therefore, INDERAL should be titrated carefully when administered for arrhythmias occurring during anesthesia.

IN PATIENTS UNDERGOING MAJOR SURGERY, beta blockade impairs the ability of the heart to respond to reflex stimuli. For this reason, with the exception of pheochromocytoma, INDERAL should be withdrawn 48 hours prior to surgery, at which time all chemical and physiologic effects are gone according to available evidence. However, in case of emergency surgery, since INDERAL is a competitive inhibitor of beta receptor agonists, its effects can be reversed by administration of such agents, *e.g.*, isoproterenol or levarterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in restarting and maintaining the heart beat has also been reported.

IN PATIENTS PRONE TO NONALLERGIC BRONCHOSPASM (*e.g.*, CHRONIC BRONCHITIS, EMPHYSEMA), INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETICS AND PATIENTS SUBJECT TO HYPOGLYCEMIA. Because of its beta-adrenergic blocking activity, INDERAL may prevent the appearance of premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia. This is especially important to keep in mind in patients with labile diabetes. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

USE IN PREGNANCY. The safe use of INDERAL in human pregnancy has not been established. Use of any drug in pregnancy or women of childbearing potential requires that the possible risk to mother and/or fetus be weighed against the expected therapeutic benefit. Embryotoxic effects have been seen in animal studies at doses about 10 times the maximum recommended human dose.

PRECAUTIONS

Patients receiving catecholamine depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine blocking action of this drug may then produce an excessive reduction of the resting sympathetic nervous activity. Occasionally, the pharmacologic activity of INDERAL may produce hypotension and/or marked bradycardia resulting in vertigo, syncopal attacks, or orthostatic hypotension.

As with any new drug given over prolonged periods, laboratory parameters should be observed at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function.

ADVERSE REACTIONS

Cardiovascular: bradycardia, congestive heart failure; intensification of AV block; hypotension, paresthesia of hands; arterial insufficiency, usually of the Raynaud type; thrombocytopenic purpura.

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Miscellaneous: reversible alopecia. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been conclusively associated with propranolol.

Clinical Laboratory Test Findings: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DOSAGE AND ADMINISTRATION

ORAL

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 40 mg INDERAL twice daily, whether used alone or added to a diuretic. Dosage may be increased gradually until adequate blood pressure is achieved. The usual dosage is 160 to 480 mg per day. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

While twice-daily dosing is effective and can maintain a reduction in blood pressure throughout the day, some patients, especially when lower doses are used, may experience a modest rise in blood pressure toward the end of the 12 hour dosing interval. This can be evaluated by measuring blood pressure near the end of the dosing interval to determine whether satisfactory control is being maintained throughout the day. If control is not adequate, a larger dose, or 3 times daily therapy may achieve better control.

PEDIATRIC DOSAGE

At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

INTRAVENOUS

The intravenous administration of INDERAL has not been evaluated adequately in the management of hypertensive emergencies.

OVERDOSAGE OR EXAGGERATED RESPONSE

IN THE EVENT OF OVERDOSAGE OR EXAGGERATED RESPONSE, THE FOLLOWING MEASURES SHOULD BE EMPLOYED:

BRADYCARDIA—ADMINISTER ATROPINE (0.25 to 1.0 mg). IF THERE IS NO RESPONSE TO VAGAL BLOCKADE, ADMINISTER ISOPROTERENOL CAUTIOUSLY.

CARDIAC FAILURE—DIGITALIZATION AND DIURETICS.

HYPOTENSION—VASOPRESSORS, *e.g.*, LEVARTERENOL OR EPINEPHRINE (THERE IS EVIDENCE THAT EPINEPHRINE IS THE DRUG OF CHOICE).

BRONCHOSPASM—ADMINISTER ISOPROTERENOL AND AMINOPHYLLINE.

HOW SUPPLIED

INDERAL (propranolol hydrochloride)

TABLETS

No. 461—Each scored tablet contains 10 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

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No. 464—Each scored tablet contains 40 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

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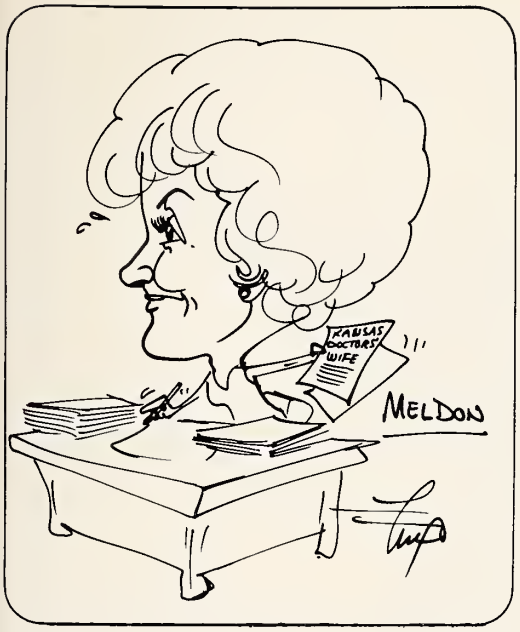
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Reference: 1. Freis, E.D. Hypertension (Suppl. II) 3:230 (Nov.-Dec.) 1981.

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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

Chairmen of Convention, Georgann Richardson and Barbara McClellan, and the Sedgwick County Auxiliary, made excellent arrangements for a comfortable and enjoyable KMSA Convention in Wichita. We're also grateful to the Jim Farhas for sharing their beautiful home for the Saturday luncheon. Thanks to many generous bidders at our auction, approximately $7,500 was collected for medical education and research. Our excellent AMAERF chairman, Carol Loeffler, did a super job in spite of having her foot in a cast!

Emerson Yoder, M.D., is serving as chairman of the Committee on Auxiliary, and has already proved his loyalty by coming up with the idea of displaying photos of KMSA past presidents in the lower level of the KMS building where pictures of KMS past presidents are displayed. He told me you voted unanimously in favor of his motion. Thank you! I know the past presidents will be pleased.

Last fall it was my privilege to hear James Sammons, M.D., speak at "Confluence" in Chicago. He showed his skill at diplomacy when he said "The greatest resource the AMA has is its Auxiliary." He said he's a strong Reagan supporter, but unfortunately the problems of medicine haven't disappeared — they've just taken a different direction. He said that instead of calling it "socialized medicine," it's now "pro-competition," and added, "Pro-competition is a new lexicon — go home and teach it to your husbands." He is certain that with the loss of federal funding, tuition in medical schools will have to go up, and some schools may close. He feels that the states should be making health care decisions.

Dr. Sammons was founder of Texas PAC and is a strong supporter of AMPAC — the political arm of the AMA. He feels AMPAC should have the support of every physician in this country and added, "It only costs the doctor $20." The law says physicians can solicit only their *members* for money, yet unions can solicit money from *anyone!* On the Equal Rights Amendment, Dr. Sammons said, "We support equality of women in all things, and have done all we can in medicine to support equality." He dislikes the punishment attitude by ERA advocates and feels that there are too many demands made. Illinois lost $150 million in convention money because of ERA boycotts.

The AMAA will celebrate its 60th anniversary at this annual meeting, June 13-16, in Chicago.

Meldon Laury
President
Kansas Medical Society Auxiliary



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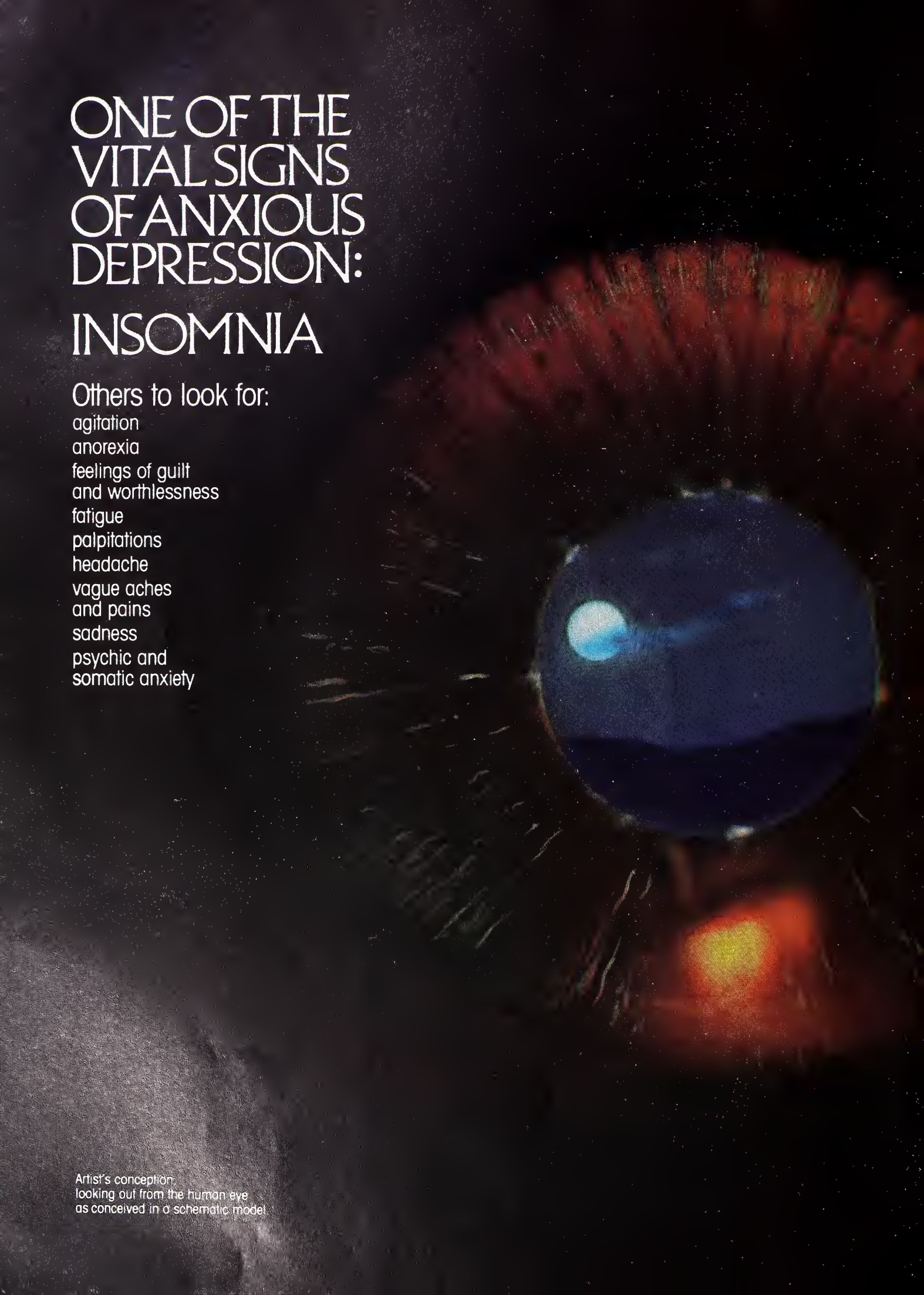
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Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use, then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10, Prescription Paks of 50.

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Official Proceedings

1982 Annual Meeting of the House of Delegates

Transactions of the 123rd Annual Session of the Kansas Medical Society are published in this issue of *The Journal*.

The resolutions are printed in numerical order under the Minutes of the Second House of Delegates Session. Those resolutions that were not adopted but were referred for further study or information are so indicated. The resolutions that failed to pass are retained in the official Minutes at the Executive Office, but are not recorded here.

FIRST SESSION

The first session of the House of Delegates of the Kansas Medical Society met on Friday, April 30, 1982, beginning at 9:00 AM, at the Hilton Inn East, Wichita. The meeting was called to order by Clair C. Conard, M.D., Speaker, who introduced Wilbur G. Cauble, M.D., President of the Medical Society of Sedgwick County.

Dr. Cauble welcomed the Delegates to Wichita on behalf of the Medical Society of Sedgwick County, and extended an invitation to all delegates to attend the activities throughout the meeting. He expressed his appreciation to Drs. Victor North, Richard A. Guthrie, and Richard J. Cummings for their efforts in planning the various activities for this annual session.

Dr. Cauble then introduced Mr. Albert Kirk, the newly elected Mayor of Wichita, who expressed his particular pleasure with addressing the House of Delegates of the Kansas Medical Society, since his brother, E. David Kirk, M.D. is a practicing physician in Wichita. The mayor expressed his appreciation to The Kansas Medical Society for scheduling the meeting in Wichita and described the various revitalization and improvement programs in the area. He urged the visitors to become better acquainted with the city and to enjoy the activities throughout the meeting.

The Speaker briefly reviewed the order of business and explained that the House was composed of elected officers, past presidents, councilors, representatives of specialty societies, and the duly elected delegates from the component medical societies. He stated that the House would follow the *Sturgis Standard Code of Parliamentary Procedure*.

Dr. Conard announced that the Reference Committee will meet after the deliberations by the House to study the resolutions. He invited all those present to appear before this committee to help draft an acceptable version of the business presented. All members of the Society are encouraged to provide input, whether delegates or not. The composition of the Reference Committee was announced as follows: Max E. Teare, M.D., Garden City, Chairman; Drs. Z. Rex Boyd, Wichita; Kenneth M. Boese, Manhattan; Dean T. Collins, Topeka; and Thomas G. Duckett II, Shawnee Mission.

Dr. Conard, Speaker, who also serves as AMA Delegate, then introduced the Officers of the Society as follows: Herman W. Hiesterman, M.D., Quinter, President; Kermit G. Wedel, M.D., Minneapolis, President Elect and AMA Alternate Delegate; Jimmie A. Gleason, M.D., Topeka, First Vice President; F. Calvin Bigler, M.D., Garden City, Second Vice President; William K. Walker, M.D., Sedan, Treasurer; Jack R. Cooper, M.D., Shawnee Mission, Constitutional Secretary; Alex Scott, M.D., Junction City, AMA Delegate; and Lew W. Purinton, M.D., Wichita, AMA Alternate Delegate.

The presence of a quorum was announced.

Upon a motion by Dr. Cooper, the Minutes of the 1981 Annual Meeting were approved by acclamation.

G. Rex Stone, M.D., Vice Speaker, appointed the following Tellers: Franklin G. Bichlmeier, M.D., Kansas City; Herbert Fransen, M.D., Newton, and David A. Leitch, M.D., Garnett.

Ballots for the primary election were distributed. There were no additional nominations from the floor and the delegates voted for the following slate:

PRESIDENT ELECT: Jimmie A. Gleason, M.D., Topeka

FIRST VICE PRESIDENT: F. Calvin Bigler, M.D., Garden City.

SECOND VICE PRESIDENT: Clair C. Conard, M.D., Dodge City; Jack R. Cooper, M.D., Shawnee Mission; Lew W. Purinton, M.D. Wichita

CONSTITUTIONAL SECRETARY: K. William Bruner, Jr., M.D., Wichita; Virginia T. Gruendel, M.D., Kansas City; Wayne O. Wallace, Jr., M.D., Atchison

TREASURER: Richard Meidinger, M.D., Topeka; William K. Walker, M.D., Sedan

AMA DELEGATE: Donald D. Goering, M.D., Coldwater; Alex Scott, M.D., Junction City

AMA Alternate Delegate: Warren E. Meyer, M.D., Wichita; and Linda D. Warren, M.D., Hanover

Speaker: G. Rex Stone, M.D., Manhattan

Vice Speaker: Franklin G. Bichlmeier, M.D., Kansas City

David E. Gray, M.D., Editor, read the Necrology Report (see below), after which the House stood for a moment of silence in tribute to the members who had died during the past year.

Dr. Gray then presented the report of the Editorial Board (see below), after which he and Dr. Hiesterman exchanged bound volumes of *The Journal*.

Jack R. Cooper, M.D., Constitutional Secretary, next presented the Report on Membership (see below).

William K. Walker, M.D., Treasurer, presented his report. He briefly referred to the printed Treasurer's Report which was distributed to the House and stated that although the Society is in sound financial condition, the Council will probably need to consider a dues increase for 1983 to maintain that position. Dr. Walker indicated that Kansas presently rates 47th in the amount of state dues out of the 52 states and territories. The House accepted the Treasurer's Report.

Presenting the Legislative Report in his new capacity as Director of Governmental Affairs of the Kansas Medical Society, Mr. Jerry Slaughter expressed his appreciation for his continued association with KMS. He reported that the Legislature adjourned this morning at 2:30 AM after passing most of the funding bills, but not a severance tax. Pending the Governor's signature, the practice of naturopathy in Kansas will become a reality with passage of HB-3144, a bill that would provide financing of osteopathic scholarships. An amendment to the bill exempts naturopaths from the Healing Arts Act and allows them to practice unrestricted in the State of Kansas. Only the Kansas Medical Society and the Department of Health and Environment opposed this issue publicly. Physicians were encouraged to contact Governor Carlin asking that he veto this bill. Another bill that passed was HB-2750, Certificate of Need, which raised the thresholds for major medical equipment to $400,000 and for capital improvements to $600,000. Another issue of considerable interest to physicians was a bill that would provide for sales tax on legal, medical, and dental services. This bill remained in committee with no action taken. Another bill introduced would require physicians to notify their patients of acceptance of Medicare assignments. This was supported by the American Association of Retired Persons and the Kansas Department on Aging. This bill was killed, but the Kansas Medical Society will work with the Department and AARP to develop an informational program to address the issue. The final Legislative Bulletin, to be mailed by the Executive Office next week, will contain the final summary of legislative activities.

Mr. Slaughter reported that he resigned as Executive Director of the Kansas Medical Society to pursue other business interests, and that the Society has agreed to retain him as its primary lobbyist. With the employment of Mr. Steven D. Carter as the new Executive Director, Mr. Slaughter foresees for the Kansas Medical Society a more effective voice in the Legislature while the administrative affairs at the Executive Office will be conducted more efficiently. He expressed his appreciation for the opportunity to serve the physicians of Kansas for the past nine years and looked forward to a continuing relationship.

Subsequently, the House adopted by acclamation Resolution 82-13, commending Mr. Slaughter for his excellent service.

Jimmie A. Gleason, M.D., Chairman of the Legislative Committee, gave his report. The Legislature passed a bill effective for one year allowing physicians in communities with no pharmacies to delegate the dispensing of medications to their nurses. This is a compromise between the Medical Society and the Pharmacy Association. The two organizations will meet during the one-year interim to work out a long-term solution. Also passed was a bill extending the life of the Health Care Stabilization Fund, which heretofore had been renewed on a periodic basis. It has now been approved to continue on a long-term basis.

As envisioned by the Legislative Committee, future problems that will concern the Kansas Medical Society will include the Certificate of Need, cost containment, the University of Kansas Medical Center and its outlying clinics, welfare, with the increasing numbers of persons becoming eligible, Medicare, block grants, and competition from greater numbers of physicians. Because this is an election year, each physician was encouraged to support candidates individually and also to become members of KaMPAC. During the coming year, the committee is planning A Day in the Legislature for physicians. An address by the Governor and a meeting of the Legislative Committee would be included in the program as a learning opportunity for physicians to familiarize them with the legislative process, the legislators, and medical issues. The Committee solicits the continued support of the members in order to remain effective in the legislative arena.

The Speaker then invited Auxiliary representatives to address the House. Isobel Dvorsky (Harry S.), San Leandro, California, President of the AMA Auxiliary, made a few brief comments (see below). She was followed by Betty L. Moore (Robert F.), Caney, President of the KMS Auxiliary (report follows).

Ivan E. Rhodes, M.D., Chairman of the Impaired Physician Committee, reported that the Committee, in an attempt to more adequately identify itself, changed the name from the Impaired Physician Program to the Committee for the Impaired Physician. Included in committee membership are representatives from the KMS Auxiliary and the School of Medicine. Included in an ex-officio capacity is a representative from the Kansas State Board of Healing Arts. There is a network of 30-40 physicians across the state who help as confronter/managers. The committee has enjoyed excellent cooperation from hospital administrators, medical staff, allied professionals, and Alcoholics Anonymous in the state. The Committee offered assistance to other organizations wishing to establish their own program on impairment among their membership. This committee has a close working relationship with the KMS Drug Utilization Review Committee and the Judicial Committee. Because of the built-in confidentiality factor in the work of this committee, a complete and totally accurate statistical report cannot be made. However, on the basis of information available to the past four years, since the inception of the program, a total of 123 initial contacts have been made. Of these, 81 concerned drug/alcohol abuse, 30 were medical (including psychiatric) disabilities, and 12 were unsubstantiated. A total of approximately 600 hours have been spent by committee members addressing these issues. There appears to be a relapse rate of approximately 20 per cent. The Auxiliary continues to be the most important link in this program. A member of the Medical School faculty serves on the Committee to provide a direct liaison to medical students.

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At the direction of the Council, this Committee studied the development of a loan program for needy physicians. This study culminated in Resolution 82-17, which is being presented to the House for adoption. After expressing appreciation to the staff for assistance in coordinating this program, Dr. Rhodes presented a plaque to George M. Penn, M.D., former member of this Committee. The plaque read: "Presented to George M. Penn, M.D., in recognition of outstanding service to his profession in pioneering the Impaired Physician Program of Kansas and serving as its first chairman. Kansas Medical Society, April 29, 1982."

The following primary election results were announced:

SECOND VICE PRESIDENT: Clair C. Conard, M.D.; Lew W. Purinton, M.D.

CONSTITUTIONAL SECRETARY: K. William Bruner, Jr., M.D.; Virginia T. Gruendel, M.D.

Franklin G. Bichlmeier, M.D., Chairman of the Judicial Committee, reported that during its first year, the Committee worked to develop organizational process and procedures. Most problems have been referred back to the county medical society for resolution. Many of the complaints concerned fees, and most problems were the result of poor communications. A number of specialty advisory committees have been apointed to investigate the merits of individual complaints when they arise within a certain specialty.

Joan Sehdev, M.D., Topeka, Chairman, reported on the status of the KMS Ad Hoc Committee on Women in Organized Medicine (see report below). Linda D. Warren, M.D., Hanover, a member of the AMA Ad Hoc Committee on Women in Organized Medicine, also presented a report (see below).

Dr. Warren, as a member of the KMS Advisory Committee to the Kansas Medical Assistants Society, then introduced Mary Klinge, RRA, CMA-A, AAMA National Trustee. Ms Klinge is employed by the St. Joseph Memorial Hospital at Larned, where she is the Director of the Department of Quality Assurance. In her introduction, Dr. Warren stated that the Kansas Medical Assistants Society, as well

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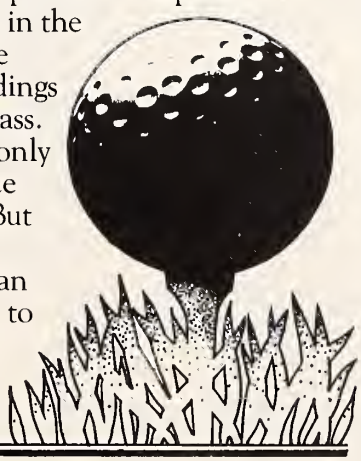
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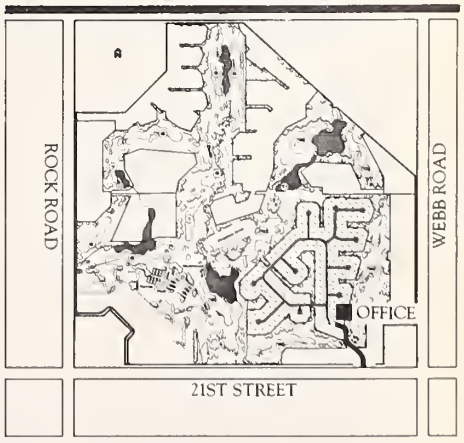
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as the American Association of Medical Assistants (AAMA), is composed of those Medical Assistants who work under the direction of a physician eligible for membership in the Kansas Medical Society and the American Medical Association. These are the receptionist, medical technologist, radiologic technologist, clerk, medical secretary, bookkeeper, registered nurse, and similar staff. AAMA was organized 25 years ago through the efforts of Kansas Medical Assistants with generous financial support from the Kansas Medical Society. Two Kansans have served as national presidents. The aim of AAMA is to provide continuing education for Medical Assistants. Ms Klinge then presented her report (see below).

Roger D. Warren, M.D., Hanover, Chairman of KaMPAC, reported next (see below). He extended an invitation to all delegates to visit the KaMPAC hospitality suite this afternoon.

The A. H. Robins Award for Community Service was presented by Mrs. Carolyn Crotty, company representative, to Carl Zacharias, M.D., Dodge City, for his outstanding contribution to the Boy Scout programs in the area.

Joseph C. Meek, Jr., M.D., Vice Chancellor for Academic Affairs, reported that the Medical School is currently experiencing a financial downturn due to a loss of patient care revenue at the Kansas City campus. The current budget is approximately $95 million, with 40 per cent coming from patient care. Dr. Meek cited three factors responsible for the current situation: (1) excessive hospital bed expansion in the Kansas City area; (2) distribution of specialists in the state; and (3) lack of marketing techniques by the Center. Dr. Meek stressed the importance of maintaining a sufficient patient load at the Medical Center because bedside education is an essential part of medical education. He invited each member of the Kansas Medical Society to consider referring at least a few patients each year to the Medical Center, rather than to referral centers outside the state. He indicated that KU will respond in kind by providing feedback information to the referring physicians. It is his opinion that a working relationship between the Medical Center and the practicing physicians in Kansas is imperative in order to maintain quality medical education in Kansas.

Robert T. Kelly, M.D., Grand Rapids, Minnesota, AMA Trustee, presented a report (see below).

Louis M. Culp, M.D., President of the Kansas Foundation for Medical Care, Inc., presented a report (see below).

In his President's Report, Herman W. Hiesterman, M.D., stated that he had visited all 19 Council Districts during this past year. He reported on the status and activity of a number of committees, including the establishment of a Member Recruitment Committee chaired by Donald D. Goering, M.D. Dr. Hiesterman also reported that a random sample survey of KMS membership has been completed and that the results are in the process of being evaluated and tabulated. The KMS Auxiliary has initiated the Public Information Program of interviewing physicians on radio spots. This project will be further developed during the coming year. The Kansas Supreme Court ruled that records maintained by the Department of Social and Rehabilitation Services concerning the provision of abortions is public information, and that the Kansas Medical Society decided not to appeal this issue. Dr. Hiesterman urged Kansas physicians to more closely cooperate with the Medical School, especially by referring patients there when appropriate.

Mr. Steven D. Carter, the newly employed Executive Director, was introduced to the House. He briefly reviewed his background, stating that his prior position was as Director of Operations for the Kansas Supreme Court. He reported that the Kansas Medical Society, as an association, enjoys an excellent reputation across the state, and that he is looking forward to the opportunity of working with the physicians. Mr. Carter also stated that in the 26 days of his new employment, he had met with 21 committees and visited four cities. He is most pleased with the continued relationship with Mr. Jerry Slaughter as Director of Governmental Affairs, and invited the Delegates to express their appreciation to Mr. Slaughter by giving a round of applause for his nine years of service.

The Speaker called the attention of the Delegates to the resolutions in the packets. All of them are to be considered automatically introduced. The rules were then suspended and the following resolutions were approved by acclamation on consent calendar:

82-13 Commendation, Mr. Jerry Slaughter

82-22 Commendation, Mr. James E. Agin

The First Session of the House of Delegates adjourned at 12:20 PM.

Necrology Committee—
David E. Gray, M.D., Chairman

Lewis Thomas, in "The Lives of a Cell," ponders our burgeoning population and inevitable death, and suggests an accommodation:

> We will have to give up the notion that death is a catastrophe, or detestable, or avoidable, or even strange. We will need to learn more about the cy-

cling of life in the rest of the system, and about our connection with the process. Everything that comes alive seems to be in trade for something that dies, cell for cell. There might be some comfort in the recognition of synchrony, in the formation that we all go down together, in the best of company.

From the Kansas Medical Society, that company includes:

Name and Address	*Age*	*Date*
Arthur J. Adams, M.D., *Wichita*	70	12/31/80
Virgil E. Brown, M.D., *Sabetha*	75	5/26/81
Homer L. Bryant, M.D., *Coffeyville*	81	12/12/80
Harold O. Bullock, M.D., *Independence*	74	5/19/81
Archie W. Butcher, M.D., *Abilene*	80	5/29/81
John S. Chen, M.D., *Hutchinson*	48	7/28/81
Robert M. Daugherty, M.D., *Meade*	72	10/30/81
Clarence A. Gilmore, M.D., *Kansas City*	50	1/1/82
Lawrence G. Heins, M.D., *Abilene*	84	5/28/81
William H. Kendal, D.O., *Gardner*	35	10/18/81
Daniel S. Klassen, M.D., *Newton*	68	10/10/81
George C. Meek, M.D., *Arkansas City*	76	3/13/82
Theodore V. Oltman, M.D., *Riley*	81	3/16/82
Louis S. Roberts, M.D., *Wichita*	85	7/8/81
James W. Shaw, M.D., *Wichita*	83	11/16/81
Robert Sohlberg, M.D., *McPherson*	75	2/22/81
Paul H. Wedin, M.D., *Wichita*	72	8/3/81
L. Barrick Wilson, M.D., *Shawnee Mission*	72	10/15/81
Lawrence E. Woodard, M.D., *Wichita*	65	3/18/81
M. Erik Wright, M.D., *Lawrence*	66	5/11/81

Editorial Board —
David E. Gray, M.D., Chairman

Again the rites of spring are celebrated across the nation. From the IRS shearing sheds, the plaintive bleatings of the shorn lambs are heard. The horn-beaked proxy flies back to its ancestral nesting place to lay its corporate eggs for the coming year. The voice of the tornado is heard throughout the land. And countless committees report to their organizations with vigor and verbosity designed to indicate great accomplishment — although not so great as to permit their termination.

In this tradition, the Editorial Board calls your attention to the healthy condition of its financial report, a product of your generosity and the Board's wisdom. The wisdom lies, as is usually the case, in having a capable staff. Nevertheless, economic attrition continues to afflict medical periodicals, and

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some continue to fall by the wayside as others constrict their frequency or format. The term, "throwaway," moves into obsolescence as publishers inform us sorrowfully that we shall now have to pay or be stricken from their mailing lists — which we didn't ask to be on in the first place. In comparison with our journalistic peers, we continue our modest but stable position, so my principal duty at this time is to render thanks to our captive subscribers.

With our financial survival assured for at least another year, we can also report that the state is generating enough illness and enough people who want to write about it to provide us with a comfortable supply of manuscripts. Publication schedules extend far enough ahead to satisfy the Board and annoy the authors who are impatient to see their work committed to the archives.

To be quite frank, then, this report has an understandable similarity to its predecessors, having the same all-too-obvious intent of encouraging your support in maintaining our financial and literary substance in the style to which we have become accustomed with the winsome acknowledgement that we are dependent on you for both.

I think you are aware that the Kansas Medical Society has been doing some soul-searching in the form of a membership survey. A sampling of the membership has been invited to register opinions about various aspects of the Society's functions and, by those mysterious machinations known only to statisticians at Ph.D. level, the innermost thoughts of the entire membership will stand revealed to our mutual benefit — or so we hope. Since one section relates to the *Journal,* the Board will be watching the results with that blend of hope and fear that accompanies the teacher's passing back the test papers. If you happen to be one of the subjects for this study, we urge you to be honest — up to a point.

You are well aware that the Supreme Court has rejected the request of the AMA to deport the Federal Trade Commission. Since the main thrust in the matter is the restraint imposed on organized medicine to control physician advertising to the laity, the *Journal* will not be greatly affected but we anticipate that increasing interest may develop in physicians' advertising to each other. In this event, we shall be considering ways and means somewhat more pur-

(Continued on page 314)

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The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

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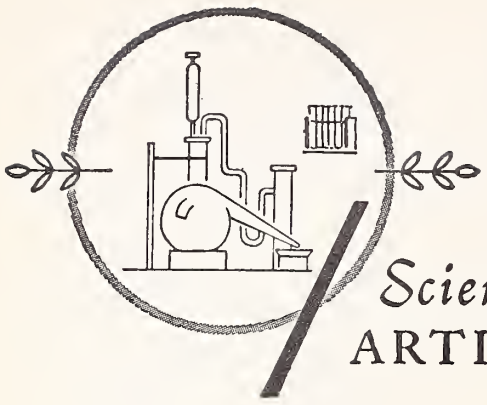
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(Continued on page 313)



Nontuberculous Mycobacterial Arthritis

Report of a Chronic Case

WILLIAM J. HALLERAN, M.D. *and* NORMAN L. MARTIN, M.D., *Kansas City, Kansas*

Case Report

A 61-YEAR-OLD Caucasian dairy farmer presented in November 1971 to the Kansas University Medical Center with swelling of the dorsum of both hands and wrists. Six months earlier his right hand had been injured without apparent skin laceration. Swelling and pain about the wrist and dorsum of the hand persisted for two months. He was placed empirically on antibiotics at another institution and experienced minimal improvement in symptoms. One month later, a fall on the left hand resulted in swelling about the distal left ulna and dorsum of the left wrist. There was no past history of family or personal tuberculosis or joint disease. Chest x-rays at presentation, and throughout his clinical course, were free of evidence of tuberculous disease. The patient did have documented essential hypertension and diet controlled adult onset diabetes mellitus.

Physical examination revealed a 6 x 3 x 2 cm non-painful, soft tissue swelling on the dorsum of the right hand, as well as a 6 x 3 x 1 cm painful, soft tissue swelling on the dorsum of the right wrist extending over the distal right radius. There was a 3 x 3 x 2 cm hot, slightly tender, erythematous soft tissue swelling overlying the dorsal aspect of the distal left ulna, and a 1 x 1 x 1 cm soft, non-tender swelling on the dorsum of the left hand. The patient remained afebrile and free of lymphadenopathy throughout his clinical course. The remainder of the physical examination was unremarkable.

Osseous and joint infections attributable to Mycobacterium intracellularis are uncommon and may or may not be associated with concurrent pulmonary or systemic infection by this organism.[1] This case illustrates manifestations of such an infection of the wrists bilaterally. A search of current literature reveals pulmonary and orthopedic references, but none in radiologic literature pertaining to osseous and joint involvement.

Results of screening laboratory blood studies, including an alkaline phosphatase test, were normal. Initial WBC was 7,000/cu mm with a normal differential. Westergren sedimentation rate was normal. An intermediate strength PPD skin test was negative, but a skin test to Type III mycobacterium was positive.

Initial radiographs of the hands and wrists, obtained in 1971, demonstrated virtually symmetric bone and joint changes *(Figure 1)*. The carpal bones show well-defined peripheral and marginal erosions with sclerotic borders and preservation of the central

From the Department of Diagnostic Radiology, The University of Kansas School of Medicine-Kansas City.

Address reprint requests to Dr. Martin, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

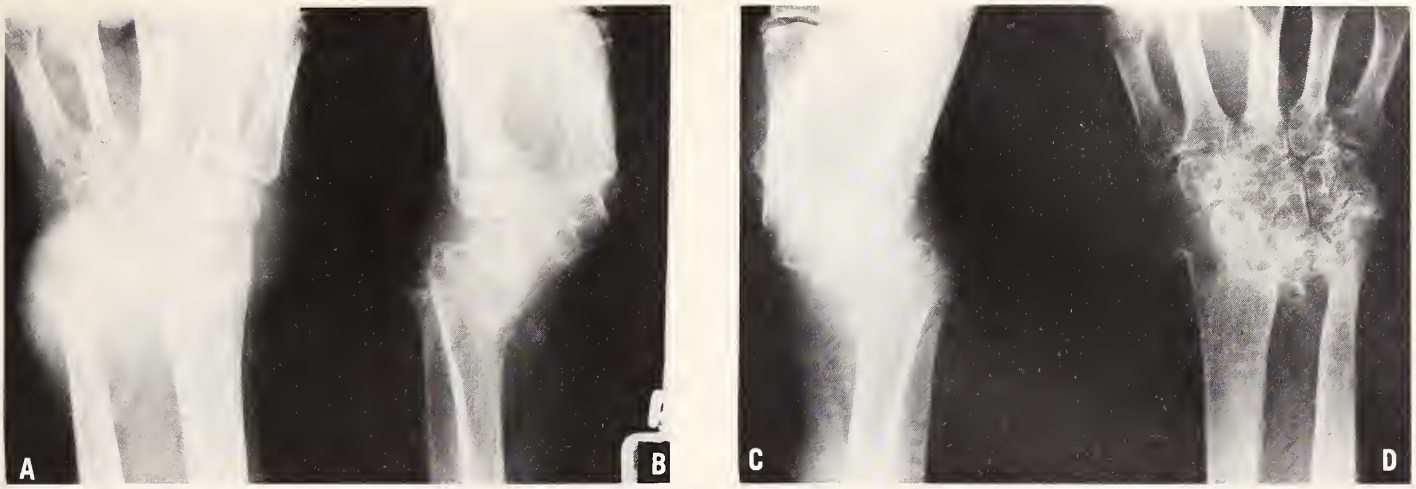


Figure 1. PA and lateral radiographs of the left (A, B) and right (C, D) wrists, dated November 1971, showing well-defined marginal erosions with sclerotic borders and preservation of the central joint space.

portion of the joint spaces. There was diffuse bony demineralization of the hands and wrists as well as soft tissue swelling over the dorsum of each wrist. There was mild palmar subluxation of the carpus.

Subsequent radiographs of both hands in 1981 demonstrate minimal progression of the destructive process of the carpal bones but with continued preservation of some of the central joint spaces *(Figure 2)*. Palmar subluxation of the carpus had progressed slightly. Diffuse bony demineralization of the hands and wrists, as well as multiple soft tissue masses were again appreciated.

Initial aspiration of joint fluid demonstrated Mycobacterium intracellularis by culture with no other bacteria or fungi present. The patient was placed on 300 mg isoniazid and 600 mg rifampin daily. Subsequent antituberculous sensitivity testing showed *in vitro* resistance of the Mycobacterium intracellularis isolate to all antituberculous drugs. The patient has experienced some improvement in symptoms and physical findings. Numerous repeated cultures of hand and wrist aspirates have been positive for Mycobacterium intracellularis. The wrist and hand soft tissue lesions have become fluctuant and frequently drain a serous fluid for extended periods of time. Antituberculous medication was discontinued after 51 months of treatment, because of inability to eradicate Mycobacterium intracellularis by culture during that time period. At no time during the past nine-year course did joint or lesion aspirate demonstrate any other pathogen by smear or culture.

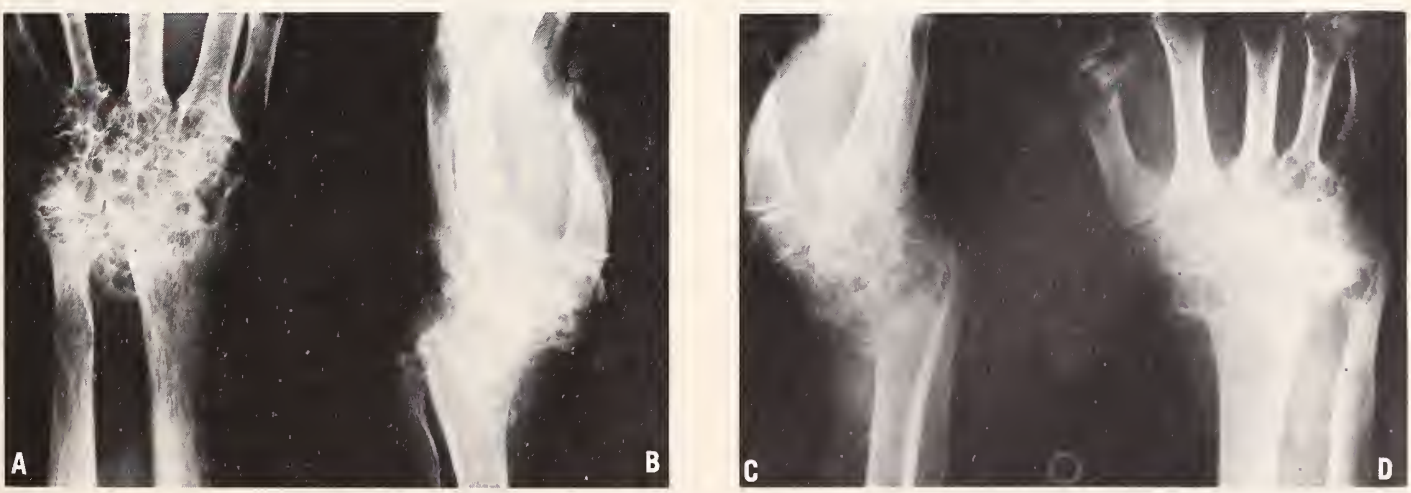


Figure 2. PA and lateral radiographs of the left (A, B) and right (C, D) wrists, dated April 1981, showing slight progression of the destructive process involving the wrists with palmar subluxation of the corpus.

Discussion

Nocardia intracellularis was first identified by Cuttino and McCabe in 1949. It was later renamed Mycobacterium intracellularis and is also known as Battey bacillus.[2] The bacillus is categorized as a Class III or slow growing potential pathogen. The colonies are white on culture, and thus non-chromogenic. Because of laboratory similarities between M. avium and M. scrofulaceum, Reznikov and Dawson coined the name MAIS complex for Mycobacterium — avium — intracellularis — scrofulaceum complex.[3] The wrists were involved with non-tuberculous Mycobacterium in three patients at the Mayo Clinic.[4] Chicoine *et al.* described a 5-year-old female with lytic lesions of all long bones, metatarsals, and metacarpals caused by M. intracellularis but with apparent arrest of the disease after two years.[5] A 14-year course of lytic lesions of the frontal bone, elbows, femora, tibiae, and ulnae secondary to M. intracellularis is cited in a personal communication by Lincoln and Gilbert.[6] In that case, it is stated that the initial presentation was fever and cervical lymph node enlargement and thereafter, successive involvement of multiple bones. At least once each year for 14 years, a bone lesion developed with associated soft tissue abscesses and drainage.

The pattern of marginal erosions with sclerotic borders and preservation of the central joint spaces seems characteristic of M. intracellularis joint infections. This appearance is secondary to the synovitis and granulation tissue that initially erodes the marginal bone and subsequently destroys cartilage. Due to lack of proteolytic enzymes which can destroy cartilage, the more central joint spaces are preserved. This has important clinical implications as patients may have continued use of the infected joints. In far advanced disease, paraosseous abscesses involve the surrounding soft tissues, and sinus tract formation develops.

Exposure to dust, animal excreta, raw milk and trauma, all of which are patient experienced, have been implicated as predisposing factors to M. intracellularis infection. This case demonstrated a documented 114 month course, with the disease presenting and remaining in both wrists without other systemic involvement and with minimal progression of the localized disease despite frequent draining soft tissue sinuses. The relative resistance of M. intracellularis to standard antituberculous regimens when compared with M. tuberculosis and M. Kansasii is quite characteristic of M. intracellularis infection. The lack of progression to other bones is somewhat unusual. The striking bilateral symmetry has not been reported to our knowledge. The usual treatment calls for surgical drainage and removal of the infected synovium and bone, which this patient would not consider, thus allowing the documentation radiographically of the 114 month course of this disease.

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Practice in Living

At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

Editor
The Journal of the Kansas Medical Society
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Diabetes I

Diabetic Ketoacidosis

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Diabetic Ketoacidosis (DKA) is a common acute complication of diabetes mellitus and constitutes a medical emergency. It is the most common reason for hospital admissions in the under-20 diabetic age group. DKA was the cause of death in one-half of all diabetics in the pre-insulin era but fortunately is responsible for less than 5 per cent of deaths in diabetics in the 1980s. Additionally, the immediate mortality from this complication was 27-44 per cent prior to 1950 but now is about 10 per cent.[1] The improved survival rate most likely results from improved understanding of the pathogenesis, disordered metabolism, and keystones to therapy of the problem. Interestingly, it is possible that most of the deaths in DKA at this time are due to complications of our therapy rather than the disease process itself.

Diabetic ketoacidosis is a common medical emergency in the insulin-dependent diabetic. Mortality from this acute complication is decreasing. Successful therapy is contingent upon understanding of the pathogenesis and pathophysiology of this condition. Frequent clinical and laboratory investigations will allow for rational choices in medical management to prevent iatrogenic catastrophes.

Pathogenesis

Excessive ketone body generation is the result of the disordered metabolism in DKA. Pathways for ketone body generation are necessary for neuronal viability, as the brain cannot use fatty acids as energy sources during times of fat breakdown and starvation as the rest of the tissues can, but the nervous system can utilize ketone bodies.

Insulin suppresses ketosis by its inhibition of peripheral lipolysis, its reduction of the ketogenic potential of the liver, and its enhancement of the peripheral utilization of ketones. In the absence of insulin, fatty acids are liberated from triglyceride stores and travel to the liver, where they are converted to their CoA derivitives. Since glycogen stores have been exhausted as sources of fuel, these derivitives are transported into the mitochondria and oxidized to acetyl CoA. Acetyl Coenzyme A (CoA) is metabolized to either adenosine triphosphate (ATP) and carbon dioxide (CO_2) by the Krebs cycle or to ketone bodies by the HMG-CoA cycle. Alterations in the fine control of the metabolism of fatty-CoA derivitives are crucial to development of DKA *(Figure 1)*.

Fatty acyl CoA can enter two pathways in the liver cell. It can either be reesterified to triglycerides or it can enter the mitochondria and be disposed of as above. The entrance of this derivitive into mitochondria is the controlling influence on this scheme and is dependent on an active carnitine acyl transferase (CAT) enzyme system at the mitochondrial membrane. Glucagon excess increases the activity of this enzyme promoting ketogenesis. This system can be depressed by increased levels of malonyl-CoA, a metabolite formed in fat synthesis from carbohydrate sources.[2] In summary, in times of positive energy balance, glucose is stored as fat turning off the carnitine acyl transferase enzyme. However, if the individual is catabolic from starvation, illness, or severe diabetes, malonyl CoA is not formed. CAT activity increases and fats are converted to CO_2, H_2O, and ketone bodies. The non-diabetic individual controls this system during periods of catabolism by using insulin to modulate lipid breakdown. The insulin-dependent diabetic loses this control mechanism.

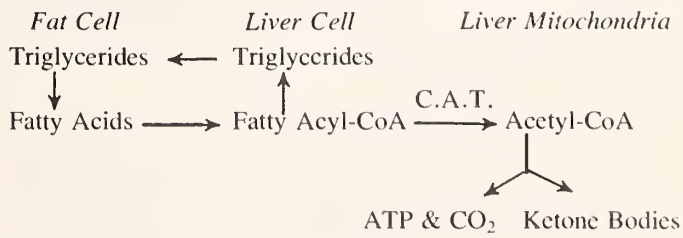


Figure 1. Profile of DKA development.

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Is total insulin lack the cause of DKA? Interestingly, most patients with DKA do have measurable amounts of insulin but the plasma levels tend to be inappropriately low for the plasma glucose. Other factors must be implicated to start the ketogenic process in motion. Stress hormones are undoubtedly important as catecholamine, glucagon, and cortisol levels are universally high in DKA. Inhibition of glucagon release with somatostatin therapy can delay the development of acidosis but not of hyperglycemia.

Diabetic ketoacidosis must therefore be viewed as a combination of relative insulin deficiency and stress hormone excess.

Precipitating Events

Infection is the trigger in 50-80 per cent of adult cases of DKA. Seemingly trivial infections, such as dental or vaginal infections, should be considered. The usual indicators of infection, such as fever and leukocytosis, may be misleading. Fever may be absent in the presence of infection and paradoxically elevated white counts can be seen in the absence of infection.[3]

Omission of insulin is the cause of 4-7 per cent of all cases in adults. Coexistent medical conditions lead to 5-10 per cent of cases. These include myocardial infarction, pancreatitis, trauma, and pregnancy. In nearly one-third of all admissions for DKA, no precipitating cause can be found.[1] It must also be remembered that many patients are newly diagnosed as being diabetic at the time of their illness, and a percentage of cases of DKA represents the initial presentation of the disease.

Presenting Symptoms and Signs

The most common presenting complaints are excessive thirst, polyuria, weight loss, nausea and vomiting, and abdominal pain. This is generally a slowly developing condition. The abdominal pain can simulate an acute abdomen in the absence of any surgical pathology.

Physical examination often reveals tachycardia, hypotension, signs of hypovolemia, Kussmaul respirations, and mild neuropsychiatric dysfunction. One quarter of patients have some mental status change, and these are correlated best with the severity of the hyperosmolar state.

Laboratory Findings

Table I lists many of the typical laboratory values in DKA. Obviously, the plasma glucose is elevated. The anion gap is increased due to the excess ketone bodies. Coexistant lactic acidosis might be present.

TABLE I
LABORATORY FINDINGS

Hyperglycemia
Metabolic acidosis
Azotemia
Ketones in urine and serum
Hypertriglyceridemia
Hyperkalemia

Patients are dehydrated resulting in azotemia. Disordered fat metabolism leads to hypertriglyceridemia.

Ketone bodies are either acetoacetate (AcAc), β-hydroxybutyrate (β-OHB), or acetone. β-OHB is the reduced form of AcAc, and is usually present in three times higher concentrations. As the acidosis increases in severity, more AcAc is converted to β-OHB. The usual method of measuring ketones — nitroprusside tablets — does not detect β-OHB. This can result in a paradoxical nitroprusside reaction; that is, the reaction can become more positive as the patient improves due to reconversion of β-OHB to AcAc. For this reason, serial acetest measurements can be misleading during therapy and need not be done after the initial determination.

Nearly all patients are total body potassium depleted by as much as 10 mEq/kg body weight. Potassium losses occur by osmotic diuresis, vomiting, and secondary hyperaldosteronism. However, plasma potassium is initially elevated due to volume contraction, extracellular shifts from intracellular stores, and tissue catabolism. Electrolytes must be monitored frequently during therapy as the serum potassium can fall precipitously once these factors resolve.

Management

Much has been written about details of the management of DKA. It is apparent that there is no one right way to manage this emergency. On the other hand, frequent clinical and laboratory examinations during the period of instability will result in the collection of a large body of important data that should be used to alter the treatment program that has been selected. Vital signs including urine output should be checked frequently. In addition, testing of plasma glucose, electrolytes, and arterial pH must be done hourly until stable.

Fluid Management

Rehydration is the first goal of therapy and is

TABLE II
FLUID MANAGEMENT

1. Begin with large volume of normal saline at the rate of 1-2 liters/hour
2. Decrease infusion rate if urine output established
3. Switch to half-normal saline once stable
4. Start dextrose containing fluids once plasma glucose reaches 250 mg/ml.

crucial to the restoration of normal metabolism. The keys to fluid management are listed in *Table II*. Large volumes of saline are used to restore blood pressure and renal blood flow. Hypotonic fluids are then employed to restore the patient's free water deficit. Dextrose containing fluids are necessary later to prevent hypoglycemia.

Potassium Replacement

The most common cause of death today from DKA is hypokalemia. It must be remembered that total body potassium is depleted and once the acidosis resolves, serum potassium can plummet. The electrocardiogram should be utilized to determine the tissue effects of the serum potassium, and continuous EKG monitoring during therapy is desirable. Institution of potassium replacement is delayed until urine output is established and there are no signs of hyperkalemia on the EKG. Patients exceedingly prone to hypokalemia are those with normal to low initial K+ levels, those who are very dehydrated, and those treated with large quantities of alkali. Usual replacement doses are 10-20 mEq K+/hour but this dose must be adjusted according to the plasma K+ level. A combination of potassium chloride and potassium phosphate may be employed.

TABLE III
INTRAVENOUS INSULIN

1. use loading dose — 0.15 U/kg regular insulin intravenously
2. give 0.1-0.2 U/kg regular insulin by continuous intravenous infusion
3. eliminate consideration of insulin binding to tubing by disposing of first 50 cc of insulin solution
4. administer rehydrating fluids by separately controlled intravenous line (''piggyback'' acceptable)
5. adjust rate of insulin dose by monitoring plasma glucose
6. start subcutaneous insulin one hour prior to discontinuing infusion

Insulin Therapy

Diabetic ketoacidosis has traditionally been treated with large doses of intravenous and subcutaneous insulin. However, during the past five to eight years, many studies have documented the efficacy of low dose insulin regimens.[4] The advantages of the low dose therapy include less hypoglycemia and less hypokalemia. It must be remembered that intravenous regular insulin lasts a very short time and hourly high-dose bolus therapy is therefore irrational. The choice of the route of insulin administration is not crucial with the exception of the avoidance of subcutaneous insulin in markedly dehydrated individuals. Protocols for low dose intravenous and intramuscular insulin are provided in *Tables III* and *IV* respectively.

Bicarbonate Therapy

Severe acidosis, indicated by an arterial pH less than 7.1, can produce nervous system alterations, left ventricular dysfunction, and hypotension. Alkali should be given to return these patients to a more physiologic pH. There are dangers in the use of bicarbonate, and these include exacerbation of hypokalemia, post-treatment alkalosis, a shift to the left in the oxyhemoglobin dissociation curve (less oxygen given up to tissues), and a paradoxical CNS acidosis. Bicarbonate should be given in small quantities, such as one ampule at a time. Once again, frequent pH determinations will help guide appropriate therapy.

Phosphate Therapy

Most patients have normal serum phosphorus levels at the time of presentation. However, within the first six hours of therapy, phosphorus levels fall precipitously. Total body phosphorus levels are low, and the inappropriate serum levels are due to extracellular shifts, as in the potassium situation. There are several consequences of hypophos-

TABLE IV
INTRAMUSCULAR INSULIN

1. do not use if patient is in shock
2. use intravenous loading dose combined with intramuscular loading using regular insulin: use 0.3-0.4 U/kg total, half intravenously and half intramuscularly
3. give 0.1-0.2 U/kg/hr regular insulin intramuscularly every hour
4. adjust insulin dose according to plasma glucose
5. switch to subcutaneous insulin when stable

TABLE V
POTASSIUM PHOSPHATE REPLACEMENT THERAPY

1. each millimole of phosphate contains 31 mg of elemental phosphorus
2. potassium phosphate solution contains 3 mmol phosphate and 4.4 mEqK + /ml
3. use 0.08-0.16 mmol phosphate/kg/dose
4. give as slow infusion
5. 5 cc of potassium phosphate contains 15 mmol phosphate and 20 mEqK +
6. EXAMPLE: a 70 kg man needs 5.6 to 11.2 mmol phosphate in first dose. A good dose would be 3 cc of potassium phosphate which would supply 9 mmol phosphate and about 12 mEqK +

phatemia, including depressed cardiac contractility, neutrophil and platelet dysfunction, confusion, and decreased red cell 2,3-DPG. This metabolite is crucial to red cell functioning, and depressed levels cause a shift to the left of the oxyhemoglobin dissociation curve (less oxygen given up to tissues).

Phosphorus replacement during ketoacidosis has therefore been popularized. The necessity to replace might be more theoretical than real as it has never been shown to hasten recovery or lower morbidity. There are risks to phosphate replacement; excessive phosphorus replacement can result in hypocalcemia and tetany.[5] Current recommendations are to avoid using phosphorus unless serum phosphorus is less than 1.0 mg/dl or to give only small quantities of phosphorus as the potassium salt. *Table V* indicates the most rational method of potassium phosphate therapy. Total body phosphorus replacement, which might need to be up to 1 mmole/kg body weight, can be done during the following week by simply feeding a balanced diet.

Summary

Diabetic ketoacidosis is still a common problem in the insulin dependent diabetic. Mortality from this acute complication is decreasing due to improved understanding of the pathogenesis, precipitating events, and therapeutic options. The important factor in determination of the above is the generation of a large body of clinical and laboratory data, and use of this information to alter therapy on a minute-to-minute basis. Absolute formulae should never be used to guide fluid, electrolytes, or insulin therapy; rather each case needs to be individualized.

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Vox Dox

Vox Dox Editor:

In the March 1982 issue of *The Journal of the Kansas Medical Society,* there is an excellent article by Stanley Friesen, M.D., entitled "Scholars, Saints, Skeletons."

However, Dr. Friesen made a statement that should be corrected. He said that "Dr. Richard L. Sutton was our first Professor of Dermatology."

Dr. William Frick was actually the first Chairman of the Department of Dermatology at UKSM, and he occupied this position from 1905-1911. From 1911 or 1912 until 1920, Dr. William McBride was Chairman of the Department. From 1916-1920, Drs. McBride and Richard L. Sutton were designated "Professors of Dermatology coordinated."

Dr. Sutton (Sr.) was Chairman of the Department from 1920-1939; followed by Charles C. Dennie, M.D. from 1939-1949; Richard L. Sutton, Jr., M.D., from 1948-1958; and then myself from 1958-1970.

In 1970, James Kalivas was appointed the first full-time non-volunteer Head of the Section of Dermatology.

This information is from year books of the early years of UKSM and the Medical School catalogs.

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Diabetes II

Can Good Control Prevent Degenerative Complications of Juvenile Diabetes Mellitus?

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CONTROVERSY regarding the need for strict metabolic control of the diabetic patient has existed for many years. A review of the statistics indicates that it is imperative that something be done to protect the Type I insulin-dependent (juvenile) diabetic from the degenerative ravages of this disease. For the patient who develops diabetes before 15 years of age, the mortality ratio is 1127 per cent greater than that of the normal population, and life expectancy is 32 years, compared to 59+ years for the non-diabetic.[1] For the poorly controlled diabetic, the mortality ratio is 2.5 times that of the well-controlled patient. The diabetic patient is twice as prone to cardiovascular and cerebrovascular arterial disease as the non-diabetic, 17 times more prone to renal disease, and 25 times more prone to blindness.[2] Many authors have concluded that good metabolic control lessens complications and that there is a correlation between the degree of control and the frequency and severity of complications. We are of this opinion and will present supporting evidence. It is acknowledged that not all investigators and diabetologists share our viewpoint. Clinical studies are presently being proposed by the National Institutes of Health to determine whether good metabolic control will affect the degenerative complications. The progression of complications worsens with poor control, and when control is relaxed, the frequency of complications increases.[3]

Blood Glucose Control

In a study of 140 patients, the degree of blood glucose was the only factor that consistently bore relationship to the incidence of vascular complications.[4] In Pirart's study, in which 22 of 23 criteria of validity were fulfilled,[5] it is conclusively proven that the diabetic neuropathy, retinopathy, and nephropathy, but not atherosclerosis, are functions of duration and severity of the diabetes and more precisely the hyperglycemia. In this study, there was a strict correlation between duration of hyperglycemia and the prevalence and incidence of neural and vascular complications. This is the only study with positive statistical proof that there is a correlation between poor control and complications. There is no role of the inherent severity of diabetes in the development of the triopathies, if the true duration and effectiveness of normoglycemia are controlled.[5] An even greater concern of this study indicates that during the first year of diabetes, hyperglycemia is more harmful than later in the disease.[5] This makes it even more critical for the newly diagnosed diabetic child to be well controlled. Closer observation of Pirart's data reveals that there is a correlation of poor control with complications in all sub-groups: male, female, family history present or absent, obesity or non-obesity, age, or severity of diabetes.[5] Diabetes mellitus initiates and is solely responsible for the microvascular complications. There does not appear to be a genetic cause for the complications.[6]

The argument about whether good metabolic control of the insulin-dependent diabetic will delay and lessen the degenerative complications still prevails. We have reviewed the pertinent literature which emphasized the value of striving for excellent metabolic control in these patients.

Our treatment goal is normoglycemia, a feeling of well-being, freedom from hypoglycemia, normal daily activities, and normal growth, development, and sexual maturation with minimal anatomical and physiological changes. In reviewing the various complications of diabetes, we would like to show that control does make a difference. We will discuss various modalities involved in the control of the insulin-dependent diabetic.

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Polyol Pathway and C-Peptide

The polyol pathway is most strictly related to hyperglycemia.[3] Sorbitol and fructose increase in direct relation to hyperglycemia. This is most important in the evolution of degenerative complications of the eye and nervous system. Clinical remission characterized by improved control is associated with partial functional recovery of beta-cell secretory activity, as assessed by C-peptide. Patients with the highest C-peptide levels have the most stable diabetes.[7]

Glycosylated Hemoglobin

Hemoglobin A-1 C is two to three times the normal value in diabetics. Hemoglobin A-1 C level is related to the average glucose level.[8] There is a correlation between the degree of hyperglycemia, hemoglobin A-1 C, and the severity of neuropathology.[3] With adequate control of blood glucose, hemoglobin A-1 C decreases toward a normal level and the hemoglobin has less oxygen-binding affinity.

Lipidemia

Sosenko and associates studied the relationship of plasma lipid levels to the degree of blood glucose control in 105 patients. Increasingly poor control was associated with increased total cholesterol, total triglyceride and lipoprotein levels, low-density lipoproteins, and very low-density lipoprotein subfractions, except for high-density lipoprotein cholesterol. Also, with hyperglycemia in uncontrolled diabetes, there is an increase in free fatty acids.[9] Three other studies have arrived at similar conclusions.

Growth and Development

Growth is also affected by diabetic control. With adequate control, growth hormone peaks decrease, a finding that is more pronounced in uncontrolled diabetics.[6] Jackson and associates studied 252 children treated twice daily with regular and lente insulins. They found that all children with good to fair-to-good control matured at a normal rate. Of more import was that all children in their group with fair control who then had good control had accelerated growth.[4] Gerstner and associates found that, with the establishment of normoglycemia via the use of a portable subcutaneous insulin infusion system, increased urinary calcium and phosphorus excretion in young insulin-dependent diabetics could be reduced.[10]

Microangiopathy

In looking at the pathology of diabetes, poor control is associated with a higher prevalence and incidence of microangiopathy.[3] Pathological changes may not be the obligatory changes of a genetic disease, but a result of the metabolic milieu in the uncontrolled diabetic. It has been shown in many animal models that hyperglycemia leads to microangiopathy. A multitude of studies have shown that metabolic change is a major cause of vascular complications.[11] In a juvenile diabetic with the disease for longer than 15 years' duration, microangiopathy is directly related to blood glucose control.[12] However, there is no correlation with control and coronary insufficiency or leg arteritis. It has also been shown that with adequate control the incidence of new microangiopathy decreases.

Capillary Basement Membrane

Of major importance is the study of the capillary basement membrane in the diabetic patient. The glomerular basement membrane glycoprotein is increased in the proportion of disaccharide units in diabetics. There is increased enzyme activity in the disaccharide units, and the hyperglycemia is associated with the increased rate of synthesis of total glomerular basement membrane. There is thickening in the capillary basement membrane in the muscle of a diabetic, and it is related to the duration of carbohydrate intolerance and to the severity of the diabetes. In discordant twins there are no capillary basement membrane changes in the non-diabetic sibling. Several studies have shown a high correlation between capillary basement membrane thickening and control of the diabetes. Only one study, by Siperstein *et al.*, showed that there was no relation between degree of blood glucose abnormality, duration of diabetes, and the width of the muscle capillary basement membrane.[8] With good metabolic control, the basement membrane thickening decreases.

Atherosclerosis

Atherosclerosis is the only complication that is unrelated to the degree of control.[5] In fact, there is a slightly increased prevalence of atherosclerosis in the well-controlled diabetic.[5]

Psychological

There are other reasons for good diabetic control. One study has shown that there is better mental and emotional health in well-controlled diabetics than in those patients who are poorly controlled.[4]

Hematology

In diabetes, there are also changes in the blood. There are increased blood viscosity,[6] increased platelet aggregation, red cell deformities, and decreased fibrinolysis. Platelets aggregate and adhere to the vessel walls, and this is postulated to be one of the reasons why there is increased atherosclerosis. In uncontrolled diabetics, the red cells have increased hemoglobin A-1 C with decreased oxygen release and increased RBC aggregation. The white cells have decreased chemotaxis and decreased phagocytosis, and the platelets again aggregate and adhere to the blood vessel walls.[6] Many of these changes are reversible with good diabetic control.

Nephropathy

In the nephropathy of diabetes, there are also many arguments for strict control. With the discovery of diabetes, there is a 1 per cent prevalence of nephropathy; after 20-25 years of the disease, there is a 15 per cent prevalence.[5] If the true duration and effectiveness of glycemia control are attained, there is no correlation between the inherent severity of the diabetes and development of nephropathy. Nephropathy is 20 times more prevalent in poorly controlled diabetics after 25 years than in those diabetics with excellent control.[5] There is a correlation between progression of the degree of proteinuria and poor control, and protein excretion can be normalized with good control.[3] Cigarette smoking increases albuminuria. With uncontrolled diabetes, there is increased capillary permeability and proteinuria, which can be reversed with good control.[6] Renal biopsies in juvenile diabetics are normal at the onset of the disease. There are slight changes even after two months of the disease, and there is more progression even after two months in the poorly controlled diabetic. Mauer's elegant studies have shown that normal kidneys transplanted into diabetics proceed to undergo diabetic degenerative changes. In two grafts from diabetic patients given to nondiabetic patients, there was regression of the degenerative renal changes of diabetes in those kidneys. He studied 12 diabetic renal grafts. Ten of the 12 developed glomerulosclerosis.[13] Renal transplantation of kidneys from diabetic to non-diabetic rats results within two months in the disappearance of IGG, IGM, and C-3 deposits in the mesangium, and there is an arrest or reversal of mesangial thickening. If one transplants normal kidneys into the diabetic, there is IGG, IGM, and C-3 deposition in the mesangium and thickening within two months.[14] In streptozotocin-induced diabetic rats given pancreatic islet cell transplant, there is decreased mesangial thickening, and IGG, IGM, and C-3 deposition is decreased in six to eight months.[15] Mauer and associates studied transplants in 12 diabetics and 17 nondiabetics. IGG staining of the glomerular basement membrane is greater in the diabetic than in the nondiabetic individual.[16] Early in the diabetes there is an increased thickness in the capillary basement membrane which is reversed after adequate control. There is strong evidence that the diabetic renal changes are due to the hyperglycemic milieu and not due to a genetic predisposition to renal disease in the diabetic patient.

Retinopathy

A multitude of studies shows that the retinopathy of diabetes is directly related to the adequacy of control. Among 41 reports, only Knowles shows no correlation between the degree of control and retinopathy.[5] Blankenship and Skyler have comprehensively reviewed the subject of diabetic retinopathy.[17] Jobe and associates showed that with poor control there was increased microangiopathy.[18] In uncontrolled diabetics there is increased capillary permeability in the retina. Retinopathy worsens in uncontrolled diabetics and is strongly related to the plasma glucose concentration. There is evidence that retinopathy decreases with strict control. There exists an association between systolic blood pressure and development of retinopathy, and an association between systolic blood pressure and the level of glucose concentration. It has also been shown that retinal permeability normalizes with good control.[19] At discovery of diabetes, 7.2 per cent of patients have retinopathy; after 20-25 years, 55 per cent of patients have retinopathy.[5] The annual incidence of retinopathy clearly and separately is related to glycemia control, and prevalence of retinopathy is clearly higher in those with chronic poor control.

An important diagnostic tool for diabetic retinopathy is fluorescein angiography. Certain investigators have indicated that a significant pathologic process is occurring at the time of onset of diabetes.[20-22] The superiority of angiography over ophthalmoscopic examination is emphasized. One group[20] could not relate the duration of diabetes with the occurrence of diabetic retinopathy by fluorescein studies. They are of the opinion that vascular changes may occur prior to metabolic changes or overt diabetes mellitus and that the vascular changes are related to the metabolic derangement, and not to the degree of control. Dorchy and associates[23] have shown that angiofluorography doubles the diagnosis frequency of diabetic retinopathy. The duration of

the diabetes markedly increases the risk for diabetic retinopathy in children.

Our personal experience with 71 diabetic children, ages 5-18 years (mean, 12.1 years), and a mean duration of diabetes of 4.8 years (range, 0.2-14 years), showed no retinopathy in 70 patients and minimal microaneurysms in one patient. These children had retinal photography, slit-lamp examination, and fluorescein retinal angiography. Of interest is that 23 per cent of these diabetic children had elevated plasma co-factor activity without any clinical correlation to the existence of retinopathy. This co-factor in the plasma of patients with diabetes mellitus potentiates the degree of aggregation by normal platelets after stimulation with low levels of ADP.[24]

Neuropathy

In the study of 4,400 patients, Pirart found a strong relationship between complications and the control of the disease. Poor control is associated with a higher prevalence and incidence of neuropathy.[5] It has also been shown that there is a correlation between the degree of glycemia control, hemoglobin A-1 C, and the severity of neuropathy. At the onset of diabetes, 7.5 per cent of patients have neuropathy; after 20-25 years, 45 per cent have neuropathy.[5] The enhanced severity of diabetes has no role in the development of neuropathy, if the true duration and effectiveness of glycemia control are maintained. With chronic poor control, there is a higher incidence of neuropathy. After 20-25 years of diabetes, the diabetic has a three times greater incidence than the normal population of neuropathy, but the poorly controlled diabetic has a six times greater incidence than the normal population.[5] Most recently, West and associates have shown, in a study of 973 diabetic subjects, that "the most powerful risk factor for microangiopathy was duration of diabetes, but frequency of both retinopathy and nephropathy was impressively related to the level of plasma glucose."[25] There is an altered metabolism in the nervous tissue, with a delay of nerve conduction velocity. Also, there is altered myelin metabolism with decreased free myoinositol in the poorly controlled diabetic.[26] The nerve conduction velocity is related to the level of myoinositol in the nerves. In the uncontrolled diabetic there are decreased nerve myoinositol, decreased nerve conduction velocity, and autonomic and motor nerve changes. When glucose concentration is controlled, there is a reversal of delayed nerve conduction velocity in diabetic neuropathy. The myoinositol changes are reversed by good control, and insulin therapy prevents impaired nerve function only when the hyperglycemia is decreased.[3] Insulin deficiency and hyperglycemia are primarily involved in pathology of impaired nerve conduction velocity.

Conclusion

In reviewing the many facets of diabetes and its complications, we have seen biochemically, in growth and development, psychologically, in blood parameters, in capillary basement membranes and microangiopathy, in nephropathy, in neuropathy, and in retinopathy that the degree of control is directly related to the prevalence and incidence of the complications. Atherosclerosis is the only complication of diabetes that does not appear to be influenced by degree of metabolic control. With this amount of overwhelming evidence, there can be no argument against striving for excellent metabolic control in the Type I diabetic patient.

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(Continued on page 333)

Mandatory CME

An Assessment by the Kansas Medical Profession

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KANSAS, in 1975, joined with other states that required continuing medical education (CME) for relicensure. Each physician was required to submit with his or her request for license renewal, evidence that the required number of hours in CME had been met. The American Medical Association's Physician Recognition Award (PRA), specialty society certificates equivalent to the PRA, and others acceptable to the AMA, satisfied this requirement. Examination of the relationship between CME credits and various privileges associated with the practice of medicine revealed no uniformity among the states regarding accreditation, reporting, or any number of factors relating to CME. To date, Kansas has not conducted any follow-up study to demonstrate the possible effectiveness of this requirement.

This study undertook to evaluate the effectiveness of mandatory CME in Kansas in terms of increased participation in CME activities and changes in the physician's approach to his practice through attendance at CME programs. To date there is no evidence to support a strong correlation between physician competence and number of CME credits earned, since the AMA/PRA accepts the number of hours submitted by the physician without evaluation of the programs attended. The ultimate responsibility for maintaining competence depends on the individual physician and not the CME programs. It has been assumed that physician attendance at CME programs will improve or at least maintain high quality medical care delivery. "While participation in continuing medical education programs should be endorsed by all, its end point should not be accumulation of credits through attendance at the programs; rather, it should be the demonstration that the care of patients is as good as possible, which is to say, it must reflect the competence of the student."[1] Neither the Kansas State Board nor the Kansas Medical Society has addressed the problem of evaluation of the Category I CME courses in relation to subject matter or instruction.

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In 1975, legislation was enacted making continuing medical education a prerequisite for relicensure in Kansas. This study was undertaken to assess the impact of mandatory CME. The results indicate that present CME requirements have neither changed the Kansas physician's approach to medical practice nor increased his/her attendance at CME programs.

Literature Review

The emphasis given to CME in recent years is evident in the number of articles that have appeared in journals and books. At the same time, a continuing debate on the effectiveness of the programs has also emerged. However, articles relating to evaluation, methodology, and variables evaluated, and the results of program evaluations are few in number. For source articles, reference was made to the *Cumulated Index Medicus* from 1965 to 1980, *Medlars II,* National Library of Medicine's National Interactive Retrieval Service, and the bibliographic sections of various journals.

Vollan[2] completed a three-year study of CME and recommended that "For CME to develop soundly there should be a clearly understood goal and specific objectives; effective leadership; more attention paid to consistent quality rather than enrollment figures; improved courses to encourage physicians not currently attending; and an expanded number of courses with more equitable geographic distribution."

Richards[3] reviewed the role of the medical school in traditional CME since this form has grown dramatically since 1960. The courses listed in the Annual Supplement of the *Journal of the American Medical Association* had grown from 1,146 in 1962-63 to 7,330 in 1977-78. Courses sponsored by

medical schools increased during the same period from 626 to more than 4,000; and although these courses account for only a portion of accredited courses, the faculty of medical schools were frequently the organizers and teachers of additional courses sponsored by specialty societies, voluntary health organizations, and state medical societies.

Methods of traditional CME have not changed since the 1930s, and as pointed out by Brown and Uhl,[4] the accreditation of CME programs and courses by the AMA achieved a special status of credibility by certifying that such activities had value for continuing medical education. These programs, however, were usually shown to produce no significant change in physician behavior or improvement in his practice of medicine.

Lewis and Hassenein[5] attempted to assess the impact of traditional CME on physician behavior. Their study suggested that the limiting factors for the outcome evaluation of CME effectiveness lie in the system of medical care, not primarily in educational or evaluative methods.

With the present system of mandatory CME for relicensure in Kansas, there is a need for assessing the criteria relating to various categories of continuing education. No conclusions have been presented in which emphasis has been placed on evaluation of CME programs. It is important that concensus be developed on the basic requirements of measuring physician performance and the overall care received by patients.

Method

The purpose of this study was to assess how physicians in Kansas participated in CME programs presently offered, and how they evaluated CME as a process that would change their approach to the practice of medicine. A random sample of the membership of the Kansas Medical Society was used in this study. A questionnaire was developed to obtain the physician's assessment of (1) the value of CME in transmitting information that would improve the practice of medicine, and (2) changes made in diagnosis, treatment, and patient follow-up as a result of CME. Code numbers were assigned to protect identity and to assist in making any needed inquiries. The list of names and code numbers was destroyed at the end of the study.

The physicians, chosen by random sampling (N = 625), received a cover letter and questionnaire. The questionnaire was divided into four sections that elicited subjective responses to questions asked, particularly in assessment of CME. A total of 528 questionnaires (83.3%) were returned, a response that can be considered a valid sampling of the Kansas Medical Society's membership and their assessment of mandatory CME. Data from the returned questionnaires were analyzed by computer. An attempt was made in the analysis of data to separate major specialty groups, although subspecialty groups responding were also coded. Results were also coded relative to type of practice, place of practice, age of respondent, hospital size, and board certification. The number of hours devoted to CME programs and courses during the previous three years and the percentage of those hours related to the respondent's specialty were determined.

Results

One hundred seventy-four physicians (33%) practiced in medical specialties, and 151 (29%) in surgical specialties. One hundred thirty-two (25%) listed family practice as their type of practice. Despite an assumption that at some point physicians would have to increase CME activity after the state board made it mandatory for relicensure, 61 per cent of those responding claimed to have continued the same pattern of compiling CME credits as they had in the past, while 39 per cent increased attendance at programs, particularly those in Category I.

Chi square tests were done on each independent variable and frequency scores for the number of hours of attendance at CME programs. There was a statistically significant variation between the number of CME hours completed and the physician's age. In the area of percentages of CME programs attended relative to the physician's specialty, a relationship was established between the percentage of CME programs in the physician's own specialty and age. None of the other variables indicated a significant variation.

Analysis of variance was used to determine if any relationship existed between the physician's perception of quality and type of practice, place of practice, age of respondent, hospital size, and board certification. On the local level, overall, physicians did perceive a difference in the quality of CME based upon type and place of practice, and hospital size. At the state level, there does not appear to be any significant variance in the perception of the quality of CME by physicians. The modality of CME does not appear to affect quality as it relates to type and place of practice, hospital size, and board certification.

Respondents were asked to rate various CME activities on a Likert five-point scale according to how useful each had been for them as a method for keeping current with medical knowledge and technology through CME. This survey showed a slight prefer-

TABLE I
MEAN SCORES* FOR PHYSICIAN'S PERCEPTION OF OVERALL QUALITY OF CME AT THE LOCAL LEVEL

CME Activities	*Mean*
Formal Programs	3.5219
Informal Programs	3.1676
C-P Conferences	3.1721
Staff Meetings	3.2535
Specialty Section Conferences	3.2754

* Likert Scale of One = Very Poor to Five = Excellent.

TABLE II
MEAN SCORES* FOR PHYSICIAN'S PERCEPTION OF OVERALL QUALITY OF CME AT THE STATE LEVEL

CME Activities	*Mean*
Medical school programs	3.7406
State medical society programs	3.2213
Specialty society programs	3.6914
Workshops/seminars in specialty subjects	3.6337
Circuit courses	3.4356

* Likert Scale of One = Very Poor to Five = Excellent.

ence among Kansas physicians for the traditional lecture format for programs at the local level *(Table I)*, and medical school programs at the state level *(Table II)*. Linear regression allowed us to predict at the .05 level of confidence the overall quality of CME available at the local and state level. In formal programs at the local level, institutional physicians saw the quality of CME to be of better quality than did physicians in solo, partnership, and group practice. Physicians who practice at hospitals with more than 401 beds saw the quality of CME to be significantly better than physicians who attend patients in hospitals with fewer than 100 beds. Hospital size was a predictor in terms of the quality of CME presented at staff meetings at the local level, and physicians saw the quality to be higher in direct proportion to the size of the hospital.

Medical school programs at the state level were rated by the family practice physician as having better quality of presentation and choice of subject matter. In Kansas, circuit courses designed for the family practitioner are presented each year by the faculty of the medical school in various small communities. It would be expected that physicians would rate the circuit courses as being the best quality of CME available in their area.

Physicians who practiced at hospitals with more than 750 beds rated teaching residents and allied health personnel as highly effective in transmitting information that would help to keep them current. Most large hospitals have training programs as well as a Director of Medical Education to develop CME programs and courses for residents, allied health personnel, and staff physicians.

Three hundred seven respondents (70%) participated in some type of evaluation of CME programs attended during a three-year period. The primary method of evaluation was a questionnaire addressing only an assessment of the program, the location, and the instructor (57%). One hundred fifty-three (27%) answered a pretest prior to the beginning of a program and a posttest after the program had been completed *(Table III)*.

The PRA was rated as the most effective method for meeting the requirement of mandatory CME for relicensure or recertification (75%). Twenty-four per cent favored their specialty society award as a method of satisfying mandatory requirements.

Discussion

The majority of Kansas physicians did not increase their participation in CME programs after the state made it mandatory for relicensure in 1975. There has been a sharp increase in CME programs as a growing number of states, including Kansas, have required documentation of CME credits. Consequently, some physicians who were previously nonparticipants are now attending CME programs to earn Category I credit, and others may be attending more frequently, since the number of programs available to Kansas physicians has risen sharply with

TABLE III

CME Evaluation by Participation	*Frequency*	*%*
Participation	370	70.342
Non-participation	155	29.468
Questionnaire-program evaluation	311	58.900
Written examination	62	11.740
Pretest — posttest	153	27.083
Pretest — posttest, Follow-up posttest	23	4.356
Posttest only	32	6.060
Observation by instructor	12	2.272
Interview	11	2.083

the result that physicians have had greater opportunity for participation. Physicians undoubtedly perceive a necessity for keeping current with new developments. They respond to CME programs designed to meet their needs and relevant to their practice of medicine.

Kansas physicians favored the traditional lecture format, apparently finding it the most efficient and effective method of knowledge transfer. Richards[3] states that "The popularity of lectures might be that physicians are simply more comfortable with them since most education from elementary school on is passive learning with an emphasis on information transfer. However, this knowledge cannot be transferred into improved patient care."

The number of hours of CME attended and the percentage devoted to the physician's specialty varies with the age of the physician. Physicians in their 50s and 60s showed the highest level of participation in CME activities. This is explained by the physician's recognition of the need for refresher courses and an adequate income for CME expenses. It is also possible that the physician in this age group is making a deliberate effort to reduce the size of his practice, thus making more time available for increased CME activities.

Evaluation of CME programs did not measure the impact on the individual physician. The majority of questionnaires related to the qualitative evaluation of the CME program attended. If measurement were limited to these superficial areas of investigation, there would be inadequate and insufficient data on which to base judgments concerning the educational value of the CME program. In this way it is possible to test at least the extent to which the program has captured the attention of those for whom it was designed. It is extremely important, however, to realize that these data are useful only if most of those in attendance provide the requested information. If the response is low (65%), generalization is unreliable.[6]

In the report by Vollan,[2] the three most frequently cited reasons for not attending CME courses were, in order of importance: (1) difficulty in obtaining coverage for practice; (2) courses given at an unsuitable time; and (3) cost involvement. In the present study, the reasons given were: (1) lack of time; (2) high expense; and (3) difficulty in getting coverage for practice. The substance of the replies was similar even though 15 years had elapsed.

This study does not answer the question of whether mandatory CME has improved the physician's participation in CME, and no conclusion can be stated relating to changes in the physician's practice of medicine that might be attributed to CME as a direct result of participation in CME. Fifty-one per cent thought that participation did change their approach to patient care, while 48 per cent did not perceive any changes in their diagnostic methods, treatment regimens, or attitudes.

Conclusions

There is widespread agreement that the only real test of the success of CME is improved quality of patient care. It is difficult to devise specific evaluation methods which can determine at the completion of a CME program whether the knowledge, skills, and techniques of the physician have improved. These observations are important to planners of future CME programs. Since gaining of CME credits is not seen as a major motivator, CME planners should not expect availability of CME credits alone to attract physicians to previously unpopular topics or formats of programs. The ultimate goal of any form of CME cannot be achieved through a voluntary or mandatory approach. The development of evaluation methods to determine the effectiveness of CME programs in improving physician competence or quality of patient care is a most important project for the 80s.

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Medical
ETHICS

David X

Economics and Medical Ethics

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David X was delivered by cesarean section on September 21, 1971. Within five seconds he was transferred to a gnotobiological environment, where he has remained to this day, more than ten years later. His speedy neonatal transfer suggests that his attending physicians feared David might be genetically blighted. Indeed they were aware that he had a 50-50 chance of suffering combined immunodeficiency disease (CID). This was because a boy born earlier into David's family was suspected of having CID after dying of Pneumocystis carinii pneumonia at age 7 months.

David came to be known as the boy in the plastic bubble. For most of his years he lived in an eight-by-ten-foot plexiglas room or one of the puptent sized chambers attached to it. A portable arrangement allowed him to divide his time between home and hospital. In 1977 his world was greatly enlarged by a modified NASA spacesuit termed a mobile biologic isolator. For the first time he was able to pet the family dogs, explore the house, and engage in the activity that gave him the greatest pleasure — spraying a garden hose at his parents and sisters.

During the years some 30 microorganisms have penetrated David's gnotobiological barriers, and ten even managed to colonize, but to date he has had no serious infectious complications. His psychological development has contributed the most surprising medical information, aside from his long survival itself, to be gleaned from his misfortune. Despite the fact that he has been deprived of all the direct human contact long thought necessary for normal development, David shows no measurable psychological impairment. On the contrary, he is happily adjusted, brighter than normal, plays well with his friends, and relates normally and lovingly with adults.

Physicians, and medicine at large, confront increasing challenges in arriving at appropriate ethical scientific and clinical judgments in dealing with infants known to have potentially lethal genetic, metabolic, or immunologic anomalies or whose continued survival is certain to be fraught with "extravagant expense," crippling disability, and often ostracism.

David's future remains problematic. The chances are now exceedingly slight that an immunologic transplantation will succeed or that he will develop immunity spontaneously. Beginning around the 35th month he began verbalizing such contingencies as "When I get out of here I will get into the car," or "When I get out of here, I will play the piano." Still he has not asked to be removed nor has he tried to break out. Indeed one of his fears in leaving the bubble for a space suit was his concern that the suit might not protect him from germs. One can only

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conjecture regarding his reactions to puberty and an adult perception of his plight.

David's case presents a host of ethical concerns. There is the matter of informed consent, both of his parents and his earlier physicians. Studies have shown that under the best of circumstances many persons cannot or will not grasp the important scientific information imparted by their physician.[1, 2] This does not appear to be true in David's case. His parents are well educated and intelligent. They understood the initial risk of CID was 25 per cent, and that this rose to 50 per cent when, in the fifth month of pregnancy, amniocentesis was performed and revealed the fetus was a male. As devout Roman Catholics the parents never considered abortion as one of their options. What we have in David's case is the not infrequent happening in ethical decision making in medicine in which cultural factors — in this case religious convictions and a strong desire for a male child — determine the final decision even in the face of formidable odds favoring a lethal genetic defect. Given what we know of human nature, perhaps this should not surprise us. The popular health columnist of a generation ago, Logan Clendening, put it this way: "Men are not going to embrace eugenics. They are going to embrace the first likely trim-figured girl with limpid eyes and flashing teeth who comes along, in spite of the fact that her germ plasm is probably reeking with hypertension, cancer, haemophilia, colour-blindness, hayfever, epilepsy and amyotrophic lateral sclerosis."[3]

Still, David's parents did not consider the possibility that he might be confined as long as ten years, reportedly because such an outcome did not figure in the thinking of his physicians. They believed he would be cured by transplantation, or develop immunity spontaneously, or die within a year or two at most. In an ultimate sense, then, David's parents could not be fully informed because his physicians failed to consider the outcome that actually developed.

David's case illustrates the difficulty in distinguishing research from treatment, and the ethical problems confronting physicians who have much to gain from a given genetic misfortune. David's project has been funded by large grants from NIH and more than 40 scientific papers have resulted from his case, according to *American Medical News* (November 21, 1977). This is not to deprecate the importance of the medical knowledge we have gained from David nor to impute ignoble motives to his attending physicians. It is only to point out that conflicts of interest can exist in such research-therapy situations, and that even the appearance of self-interest on the part of the physicians must be scrupulously avoided.

A new ethical and legal aspect of medicine enlightened by David's case is the concept of wrongful birth. Several cases have now reached the courts in which children, upon reaching maturity, have sued their parents for giving birth or permitting medical intervention when they knew their child would be handicapped in some way. It has been reported that, in Illinois, one such case was brought merely because the child in question was a bastard. It is difficult to see the liability here, since our experience indicates that bastards often rise to high places.

A thorough discussion of this matter would take us too far afield, but it does present an intriguing new aspect to the abortion issue. Many now argue that abortion is justified for no reason beyond the mother's decision that the child is unwanted. Given a situation in which a severely defective child is predictable during pregnancy, one may now ask if there may not be a legitimate fetal right not to be born.

Perhaps the most stark ethical issue raised by David's case is economic. Would his physicians pull all stops again, faced with another David X? One is quoted in *American Medical News* as saying, "Absolutely. If we had it all to do over again, would we go to the moon again?" The other principal physician is less certain. "I would probably, if I had the facilities . . . I just really don't know what I'd do. It's such a huge endeavor from all standpoints, and such a very expensive endeavor." David's medical expenses run to $200,000 a year. The development of his mobile isolator cost $20,000 and another $2,000 will be required each time he outgrows a suit. By this time we may assume a direct outlay of more than $2,000,000 with no end in sight. Obviously few persons would suggest that David should be abandoned at this point. Still all the ethical lessons possible must be extracted from his case, and one of these is the tremendous economic implications of the first commitment in any life-saving medical situation, whether it be closing the sac in a myelomeningocele or placing an eighty-year-old stroke victim on a respirator. The first decision is almost always more than an initial commitment. If it favors life, it may commit all concerned to continued support even after the quality of life is perceived as frankly dismal.

It can be argued that saving future Davids is a small matter in a nation as wealthy as ours. After all, there are only some 200 cases of CID annually in the United States, and rarely will one be diagnosed ante-

natally. Sir McFarlane Burnet, the noted biologist, would say no even to the rare cases such as David's. "The application of science," he wrote, "to treat any child with a genetic, metabolic, or immunologic anomaly which is potentially lethal, at the best level possible in the light of current knowledge will always be extravagantly expensive. It will require a small full-time team of biochemists and technicians and will subject the child to constant examinations, blood tests and injections. In most cases such detailed control must be continued throughout life. It is a brutal fact that except in a research center in an affluent country and only when an individual scientist has a professional reputation to make or to maintain in the relevant field, such children can rarely receive empirical care and will die at the first crisis in their condition."[4]

Just what is meant by Sir McFarlane's phrase "extravagantly expensive" is better understood considering a more common neonatal defect such as myelomeningocele, which occurs once in each 500-600 births. In England and Wales for the year 1968 Lightowler calculated the incidence of myelomeningocele as 1,207. Of these some 930 would need treatment for two years and 500 would require medical attention beyond two years. Utilizing the known incidence of required operations, length of hospital stay, and other known medical costs, he calculated the cost of treating these patients at £ 1.430 million. The figures did not include costs of patients over the age of 5 years in terms of special education and hospitalization. The total rough estimate came to £ 3.75 million for a single year's group of patients with myelomeningocele. With the British pound then worth $2.40 that translated to nine million 1968 dollars.[5]

It is clear that economics and ethics are becoming increasingly entwined on the current scene. This is abhorrent to many health professionals who would prefer to go all out to save every life and let others worry about the costs. It is all but certain that this luxury which the profession enjoyed in the past cannot continue indefinitely in the face of medical costs that now account for more than 10 per cent of the gross national product. Some would avoid distasteful economics and hide behind other ethical considerations such as the quality of life. The victim of myelomeningocele, after all has been described as a paraplegic incontinent cripple, who may be snatched from the jaws of death in the neonatal months, only to be delivered over to the tragedy of an expanding head, or to a life of social exclusion amid the fumes and filth of a double incontinence and trophic ulcers. Even if this does not apply to all such persons, it does to many. Yet, if economic factors are to operate in these decisions, they should be faced squarely. In the case of myelomeningocele, costs almost certainly have figured into British practices of increasingly rigorous triage, which is also now utilized in the United States.[6]

In philosophical terms the issue hinges on what is called distributive justice, the right of each citizen to a fair distribution of society's protection and resources, unless solid ground exists for treating him differently. The principle assumes great practical significance where resources are limited, as those of medicine are. Thus some would approve expending tax monies to defray medical expenses in cases of unpredictable genetic catastropes, but might balk when the odds of a serious genetic defect are known to be 50-50 or more. Or, to return to David's specific case, would his parents have a moral claim on society to underwrite another such gamble if they decided David should have a brother? It appears that lines will have to be drawn in these matters, and they will be tough to draw. While we await some latter-day Solomon, physicians increasingly will have to consider the economics of saving lives, as odious as most will find it. It has been claimed that every time a physician puts a pen to a prescription he is engaging an ethical act. Certainly the physician plays a major role in the "first commitments" which are often long term in nature. The public will be better served by wise physician intervention in these matters than by a federal mandate allocating each physician to dispense X dollars of medical care each year, after which expenditure he retires until the next fiscal year.

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Current COMMENT

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Nail Diseases

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MOST PHYSICIANS' knowledge about nail diseases is scanty, even though they will frequently encounter these problems. The nails are an appendage of the skin, and so, like the skin, may provide useful clues to systemic disease. Nail dystrophies that are not markers of underlying disease are commonly encountered; so an understanding of nail disorders is helpful to the primary care physician as well as to dermatologists.

Anatomy

The nail unit is divided into four parts: The *proximal nail fold;* the *nail matrix;* the *nail bed;* and the *hyponychium*. Each of these parts contains an epidermis and dermis and keratinizes but it is the keratinization of the nail matrix that is now thought to be responsible for the nail plate *(Figure 1)*. The nail plate then is the stratum corneum (the uppermost layer of the epidermis) of the nail matrix. Perhaps about one cell layer of the stratum corneum of the nail bed rides along the ventral aspect of the nail plate. Hyponychium is sometimes used to refer to the entire epidermis of the nail bed but is usually used to refer just to the extension of the epidermis of the floor of the nail fold to the undersurface of the nail plate.

The proximal portion of the nail matrix is responsible for the dorsal aspect of the nail plate; the distal portion of the nail matrix (called the lunula or half moon) is responsible for the ventral aspect of the nail plate. Thus, an abnormality such as superficial pitting of the nail plate, commonly seen in psoriasis, results from a defective keratinization of the proximal nail matrix.

Eponychium is the term used to describe the anterior extension of the roof of the nail fold onto the nail plate. It consists of epidermis. The cuticle is the extension of the epidermis of the eponychium (usually just the uppermost layer of the epidermis, called the stratum corneum) over the proximal nail plate. The lunula or half moon is the white, crescent-shaped area just distal to the cuticle. It may not be visible in all people. Since it is the distal portion of the nail matrix, injury to it may cause a permanent nail dystrophy. The white color of the lunula is probably due to the looseness of the nail plate and the incomplete keratinization of the nail plate in this area.

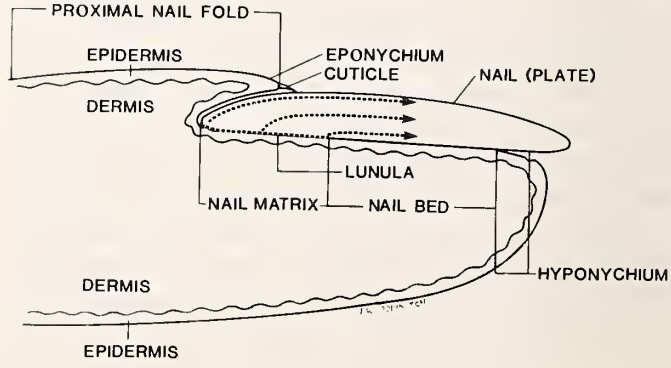


Figure 1. Sagittal section of a normal fingertip (artist's rendition).

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Calcium, which is often assumed to account for the hardness of the nail plate, probably is inconsequential, since it accounts for only 0.1-0.2 per cent of the nail plate by weight and does not differ in concentration in brittle and normal nails. The hardness of the nail plate, according to Norton,[1] is attributed to: (1) high sulfur content in the form of cystine; (2) poor ability to hold water because of low lipid content; (3) filaments aligned parallel to the nail and at right angles to the growth axis; (4) precipitation of a proteinaceous material on the cytoplasmic side of the cell membrane of the nail plate cell, forming a "marginal band"; and (5) highly developed junctional structures between the nail plate cells.

Nail Growth

Fingernails grow at a rate of 0.5-1.2 mm/week. Toenails grow at about one-half to one-third the rate of fingernails. It takes an average of 5½ months to grow a new fingernail and 12-18 months to grow a new toenail. Growth rates show a variation among individuals as well as a variation between nails on the same individual. Middle fingernails grow most rapidly; the thumb and fifth fingernails grow most slowly. Fingernails on the right hand grow more rapidly than those on the left hand. Factors such as old age, lymphedema, and many general medical illnesses may decrease nail growth rates. Nail biting, psoriasis, and many inflammatory changes around the nail may speed up nail growth.

Among the more common nail diseases are chronic paronychia, onychomycosis (tinea unguium), changes caused by trauma, psoriasis, and impaired circulation. Some nail disorders may be a sign of underlying disease; there are some disorders confined just to nails. A glossary of commonly used terms for nail dystrophies is given at the end of this article.

Nail Dystrophies

Paronychia (păr-ō-nik′ĭ-ah) is an acute or chronic inflammation of the tissues surrounding the nail plate. Chronic paronychia is caused by *Candida albicans* in about 80 per cent of cases *(Table I)*. Housewives, bakers, bartenders, dishwashers, beauticians, and others whose hands are exposed to water for long periods are among those most likely to be plagued with this problem. Vigorous manicuring may interfere with the adherence of the cuticle to the nail plate and introduce yeasts under the proximal nail fold. A contact dermatitis to food products or artificial nails, or an infestation such as scabies or chiggers, may provide the initial entry for yeasts or bacteria to establish residence under the proximal or lateral nail folds. Although candidiasis heads the list of infections causing paronychia, staphylococcal infections, herpes simplex, and syphilis are among other infections to be considered.

TABLE I
COMMON CAUSES OF NAIL DYSTROPHIES

Dystrophy	*Cause*
paronychia	Candida
onychomycosis	dermatophyte (tinea)
onychauxis	trauma fungal infection decreased peripheral circulation
onycholysis	trauma fungal infection
onychorrhexis	aging decreased peripheral circulation rheumatoid arthritis
brittle nails	aging poor peripheral circulation chemical exposure anemia
koilonychia	iron deficiency
pitting	psoriasis

It is important in treating paronychia that the patient keep the nail dry and away from chemical or physical irritants. For those who cannot avoid constant water exposure or trauma, a nail polish sealer — acting as an artificial cuticle — may be considered. An appropriate antimicrobial should be given pending culture results. Topical nystatin, miconazole, or clotrimazole are usually effective for treatment of Candida infections. In cases refractory to topical anti-yeast preparations, oral ketoconazole may be helpful. Oral griseofulvin is ineffective for candidiasis of the nails. Diabetes mellitus, acrodermatitis enteropathica (zinc deficiency disease), and muco-cutaneous candidiasis are among disorders to consider in extremely recalcitrant cases of Candida paronychia.

Onychomycosis (ŏn-e-kō-mī-ko′sis) is a fungal infection of the nail unit caused by fungi such as dermatophytes (often referred to as tinea), saprophytes, or yeasts. Clinically, onychomycosis may cause a thickening of the nail, crumbling, discoloration, or just superficial white spots. Yeasts typically involve proximal and lateral nail folds and cause a paronychia, as well as involving the nail plates. Dermatophytes and saprophytes usually involve just

the nail plate. Only recently has it been appreciated that saprophytes such as Scopulariopsis, Aspergillus, and Hendersonulla, usually thought to be nonpathogenic, may cause nail dystrophies. These saprophytes usually attack a traumatized nail. The saprophytes and yeasts do not respond to oral griseofulvin like the dermatophytes. Oral ketoconazole, which has recently been introduced on the market, is effective for both yeast and dermatophyte infections of the nail. Oral griseofulvin is considered the drug of choice for dermatophyte infections of the nail. Ketoconazole has not been formally approved for use in treating dermatophyte nail infections, but should be considered in those who either do not respond to griseofulvin or are allergic to it.

Onychauxis (ŏn-e-kawk′sis) means hypertrophy of the nail. Probably one of the more common causes of this condition is trauma to the nail *(Table I)*. It used to be quite common on the toenails of men who cared for horses, and was sometimes referred to as "hostler's toe." Fungal infection (onychomycosis), as well as peripheral neuritis, decreased peripheral circulation, thyroid disease, hemiplegia, tabes, psoriasis, ichthyosis, and leprosy may all be causes. Cases of familial onychauxis have been reported. Sometimes the nail, rather than being just thickened, will assume a claw-like appearance caused by more rapid growth of the nail matrix on one side than on the other. This claw-like condition is called *onychogryposis* (ŏn-e-kō gri-pō′sis). The causes of this condition are the same as those of onychauxis.

The treatment of hypertrophied nails is correction of the underlying cause. Sometimes nails need to be surgically removed or filed down. Massaging with warm olive oil or other emollients may be helpful. There are some chemical agents that will cause a decrease in the thickness of the nail and can be used. Forty per cent salicylic acid plasters or 40 per cent urea are keratolytic agents and will eventually dissolve the nail, but are to be used with great caution, if at all, in people with diabetes mellitus or decreased peripheral sensation. These agents may be left on the nail from 12 hours to one week. It is important that they be applied only to the nail plate and do not touch the skin surrounding the nail plate since they will cause excessive irritation. Topical urea has been used in Russia; its effectiveness has only recently come to the attention of English-speaking dermatologists. A urea formula that may be used for chemical avulsion of the nail plate is as follows: urea 40%, 120 gm; white beeswax 5%, 15 gm; anhydrous lanolin 20%, 60 gm; white petrolatum 25%, 75 gm; silica gel type H 10%, 30 gm.[2]

Onycholysis (ŏn-e-kol′ĭ sis) refers to separation of the nail plate from the nail bed starting at the distal edge. Common causes of onycholysis are trauma and fungal infections *(Table I)*. Trauma may result from over-manicuring the nails and exposure to caustic chemicals such as bleaches, weed killers, acids, or alkaline solutions which tend to loosen the attachment of the nail plate to the nail bed. Nail base coats and hardeners also may cause this problem. Candida is a more likely cause of onycholysis than the dermatophytes.

Onycholysis may be a sign of a number of systemic diseases including hypothyroidism, hyperthyroidism (often called Plummer's nails), drug ingestion, porphyrias, Raynaud's disease, syphilis, and anemia. Photosensitivity or the precipitation of porphyria by a drug may lead to onycholysis. Among the drugs that can cause onycholysis are tetracycline, topical or systemic 5-fluorouracil, and oral contraceptives. There have been some cases of hereditary onycholysis. Certain skin diseases, such as eczema, lichen planus or psoriasis, can lead to this problem as well.

Onychorrhexis (ŏn-e-kō-rex′is) refers to excessive longitudinal striation of the nail plate. Ridging of the nails is seen with old age, rheumatoid arthritis, decreased peripheral circulation, acquired hyperparathyroidism, homocystinuria, and gout. Skin diseases such as psoriasis, lichen planus, Darier's disease, and alopecia areata may cause ridges in the nails.

Brittleness of the nails may be a senile change and is seen in up to 8 per cent of normal people. Chemical exposure, especially to alkalis, can lead to this problem. Brittle nails can result from iron deficiency, poor peripheral circulation, gout, syphilis, neurological diseases and argininosuccinic aciduria, as well as skin conditions such as eczema, psoriasis, lichen planus, and leprosy.

Treatment of brittle nails includes avoidance of harsh chemicals, avoidance of frequent polishing and nail polish removing (which dry out the nails), and the discovery of an underlying cause if there is one. Frequently a night cream may help. Samman[3] recommends 2% salicylic acid ointment with equal parts of glycerin of starch applied nightly; and Pardo-Castello[4] recommends white wax 1.0 gm, lanolin 10.0 gm, with oil of sweet almonds 10 ml to be applied nightly. Oral gelatin has had mixed results in treatment of brittle nails.

There is an entity called "20 nail dystrophy" in which all 20 nails develop not only a ridging but a roughness and lackluster appearance. This condition is usually seen in otherwise normal children and does not seem to be associated with systemic dis-

ease. Most people feel that it is a localized psoriasis or lichen planus involving just the nails. Similar changes may be seen in patients with alopecia areata.

Koilonychia (koy′-lō-nik′ ĭ ah) (spoon nail) is a malformation of the nail in which the outer surface is concave. It is most commonly associated with iron deficiency anemia and may be part of the Plummer-Vinson syndrome. Sometimes patients with iron deficiency, without an obvious anemia, will have koilonychia. It has been observed in up to 50 per cent of patients with idiopathic hemochromatosis and may be seen in hyperthyroidism and hypothyroidism, polycythemia vera, trauma, coronary disease, syphilis, and Raynaud's disease. It may be associated with the skin diseases lichen planus, psoriasis, and alopecia areata. It may be a congenital anomaly (usually inherited). Koilonychia may be seen in normal infants, but usually resolves spontaneously. Trauma-induced koilonychia is commonly seen in the toenails of ricksha boys in South Africa and the fingernails of motor mechanics.

Pitting of the nails is most commonly caused by psoriasis, but different kinds of dermatitis, alopecia areata, and fungal infections also cause pitting. Minor pitting is seen in normal people. An abnormal retention of nucleii (parakeratosis) in some epidermal cells on parts of the dorsal surface of the nail plate causes these areas to shed, and leaves small indentations or pits on the surface of the nail plate.

Markers of Systemic Disease

A number of nail dystrophies discussed above may be markers of underlying disease. Certain organic diseases have nail dystrophies that are characteristic *(Table II)*.

Decreased peripheral circulation from any cause can lead to impaired nail growth, onycholysis, onychauxis, shedding of the nails (onychomadesis), pterygium formation, pterygium inversum unguis, or Beau's lines. Pterygium is an overgrowth of the cuticle onto the nail plate, giving a wing-like appearance. Pterygium inversum unguis is an abnormal attachment of the distal nail bed to the ventral surface of the nail plate. Because of decreased peripheral circulation and loss of tissue of the distal digit, it gives the appearance of several millimeters of skin without subcutaneous tissue attached to the ventral aspect of the nail plate. Systemic scleroderma may result in this deformity.

Congestive heart failure may cause a reddish discoloration of the lunulae or a striking yellow color of the nail plates. Yellow nails may be seen in edematous extremities and may be associated with bronchitis, bronchiectasis, pleural effusion, thyroid disease, or underlying neoplasm.

TABLE II
NAIL ABNORMALITIES ASSOCIATED WITH SYSTEMIC DISEASE

Systemic Disorder	*Nail Abnormalities*
decreased peripheral circulation	decreased nail growth onychauxis onychorrhexis brittle nails pterygium
congestive heart failure	yellow nails reddish lunulae
pulmonary disease	clubbing
hepatic disease	Terry's nails Muehrcke's lines blue lunulae (Wilson's disease)
renal disease	half-and-half nails
thyroid disease	onycholysis

Pulmonary disease may, in addition to yellowing, lead to clubbing of the nails. Although about 80 per cent of clubbing of the nails is associated with pulmonary problems, cardiovascular disease, thyroid disease, hepatic disease, bowel disease, and syphilis are among other causes. In some cases clubbing is inherited as an autosomal dominant trait with varying penetrance and without associated systemic disease.

Hepatic cirrhosis often causes a white discoloration of the nail bed and flattening of the nail. Terry described a whitish discoloration involving all but the distal 1-2 mm of the nail in 82 of 100 cirrhotics (''Terry's nails'').[5] Paired white transverse bands that run parallel to the lunula are seen in many cirrhotics and can be seen in anyone with very low serum albumin. These white bands are in the nail bed, do not grow out with the nail plate, and are referred to as Muehrcke's lines. A striking blue discoloration of the lunulae may be a feature of Wilson's disease.[6] Argyria and phenolphthaleins can cause a similar bluish discoloration.

Lindsay noted that renal insufficiency can cause a proximal, whitish discoloration and distal brownish color (''half and half nails'').[7] Brown nail bed arcs are another manifestation of renal disease. Nonspecific signs of bodily insult such as Beau's lines and Mees' lines may be seen in renal patients. Beau's lines are transverse grooves of the nails that

grow out with the nail. Mees' lines are transverse white lines that grow out with the nail plate and are classically associated with arsenic poisoning, but may also be seen with many other diseases including renal failure.

From this brief overview one can see that nails are subject to a variety of diseases. These include many systemic illnesses as well as strictly localized and cutaneous diseases. One should note that syphilis appears in the differential diagnoses of many nail dystrophies and should be ruled out in any nail disorder of unclear etiology.

Glossary

paronychia (păr-ō-nik′ĭ-ah)
inflammation of tissues surrounding the nail plate
onychomycosis (ŏn-e-kō-mī-kō′sis)
fungal infection of the nail plate (also called tinea unguium)
onychauxis (ŏn-e-kawk′sis)
hypertrophy of the nail plate
onychogryposis (ŏn-e-kō gri-pō′sis)
thickened, claw-like deformity of the nail
onycholysis (ŏn-e-kol′i sis)
distal separation of the nail plate from the nail bed
onychorrhexis (ŏn-e-kō-rex′is)
excessive longitudinal ridging of nail plate
koilonychia (koý-lō-nik′i ah)
concave outer surface of nail plate (''spoon nails'')
onychomadesis (on ik-ō-med-ē′sis)
shedding of the nail plate starting at the base

Self-Assessment Questions

1. A serology (VDRL) should be done in most patients with nail dystrophies. (T or F)
2. Oral griseofulvin is useful for Candida infection of the nails. (T or F)
3. Onycholysis always indicates thyroid diseases. (T or F)
4. Koilonychia is pathognomonic of iron deficiency. (T or F)
5. Nail biting retards growth of fingernails. (T or F)

(Answers on page 336)

Council Meeting

(Continued from page 267)

would be asked to supply names of physicians who could assist the Judicial Committee.

The Council reappointed David E. Gray, M.D., as Editor of *The Journal*. Also reappointed was Richard H. Greer, M.D., to an additional three-year term on the Editorial Board.

On the subject of physician advertising in *The Journal*, and after considerable discussion, the Council noted opposition to the general concept of physician advertising. Based on that discussion, the Council favored only announcements of names and addresses of new KMS members on a limited basis, with specific guidelines to be developed by the Editorial Board.

The Council reviewed those actions of the House of Delegates that required attention of the Council, specifically:

Resolution 82-3: To appoint a committee to study the Medical Examiner system; and

Resolution 82-20: To appoint a committee to study the funding of local health departments.

The Council approved a one-year agreement with Mr. Jerry Slaughter of Kansas Management Services, Inc., beginning May 1, 1982. Mr. Slaughter will be providing legislative/governmental liaison services to the Kansas Medical Society.

A report was given concerning the possibility of establishing a physician-owned professional liability insurance program in conjunction with a similar program in Oklahoma. Further consideration of this matter will be continued through the Commission on Professional Liability.

A legislative update included the discussion of SB-783, the bill relating to the dispensing physicians. Mr. Slaughter explained that a compromise had been reached which allows physicians to delegate the dispensing of drugs to nurses only in cities where no pharmacies are located. Additionally, a study committee would be appointed this summer to work with the Kansas Pharmacists Association to determine a long term solution to the problem.

The Council approved a loan to MEDISERVE, Inc. of $12,500 for the balance of 1982.

The meeting adjourned at 12:55 PM.



Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Acyclovir

Question: Could you provide information on the new antiviral agent being used in the treatment of herpesvirus infections?

Answer: Effective drug therapy of viral infections has always been limited by the toxicity and minimal spectrum of availabile agents. Acyclovir (Zovirax) — a new antiviral agent of low toxicity and high specificity against herpesvirus — shows promise of effectiveness.

Acyclovir, an analogue of guanosine, acts as an antimetabolite. The viral enzyme thymidine kinase phosphorylates acyclovir to the monophosphate form. Monophosphorylated acyclovir is then phosphorylated by other kinases to its active form, acycloguanisone triphosphate, which selectively inhibits viral DNA polymerase. It has been observed that acyclovir inhibits viral DNA polymerase 10-30 times more effectively than human polymerase, and this apparently results in specificity and low toxicity.[1]

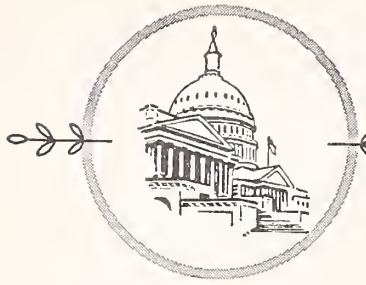
Acyclovir appears to be effective in the treatment of herpes infection (herpes simplex, herpes zoster, and varicella zoster) in immunosuppressed patients. During trials, this agent was responsible for a reduction in viral shedding, a somewhat faster resolution of pain, and healing of mucocutaneous herpetic lesions. These patients were treated with intravenous doses of 250 mg/m^2 or 3.5-5.0 mg/kg every eight hours.[2, 3] This regimen has also demonstrated efficacy in the prophylaxis of herpetic infections in immunosuppressed patients.[4]

Clinical trials have shown that acyclovir 3 per cent ophthalmic ointment administered five times daily may be effective in treating ocular herpes infections.[5, 6] Intravenous acyclovir (5 mg/kg/8 hrs for 15 doses) has also been studied in the treatment of severe primary genital herpes.[7] The medians for healing time, duration of vesicles, new lesion formation, viral shedding, and all symptoms were significantly shorter in patients treated with acyclovir than those treated with placebo (mannitol). Trials with topical acyclovir for treatment of genital herpes has produced similar results. Although this agent decreases the length of recurrent HSV attacks, it does not alter the rate of recurrences or eliminate the virus' latent phase.

To date, acyclovir has demonstrated minimal toxicity. Approximately 45-73 per cent of an intravenous dose is excreted unchanged in the urine. There have been reports of increases in blood urea nitrogen and serum creatinine during intravenous therapy, but no significant renal toxicity has been observed.[3, 9] Because acyclovir is excreted primarily by the kidneys, reduced doses may be warranted in patients with renal impairment.[10, 12]

The most dramatic effect demonstrated in initial trials is the reduction of viral shedding in all groups of patients studied. Because of its selectivity and low toxicity, acyclovir may assume a major role in the mangement of herpetic infections. Zovirax was released by Burroughs Wellcome Company in the spring of 1982. Initially it will be available in topical and injectable dosage forms.

Submitted by
Sally Koym, R.Ph.



Socio- ECONOMICS

Pegboard Power — Subduing the Paper Dragon

PAPERWORK is often the bane of medical office routine. Tax complexities, insurance procedures, and government regulations require complete, accurate, and retrievable records. Maintenance of these records with a minimum expenditure of staff time promotes economic efficiency and thus helps to control health care costs.

How can this be achieved? One popular method is the pegboard system utilizing the superbill.

The superbill is a system of forms designed to make one entry do the work of many *(Figure 1)*. Most entries are made "as-you-go" — that is, as each patient is seen — thereby virtually eliminating the old bug-a-boo of "catching up on paperwork."

Recommended primarily for solo and small group practices, the superbill system, appropriately utilized, can provide much the same analytic data as a computer at a fraction of the cost.

What are the benefits to be derived from implementing the superbill system?

- Staff time for paper work is significantly reduced.
- Increased accuracy is assured by elimination of the need to transfer information from one place to another.
- Accessibility and currency of records facilitates collections and helps to keep receivables to a minimum.
- Individual patient records can be photocopied for billing purposes, eliminating the necessity for retyping bills; the daily tote of receipts provides a simultaneous deposit slip.
- Standard forms acceptable to insurance companies can be incorporated into the system.
- A few minutes spent summarizing data at the end of each day yield a comprehensive and accurate picture of each day's business. Errors can be easily identified and corrected, and cash flow and accounts receivable data are kept up-to-the-minute.

The system is flexible and forms are adapted to meet the needs of each office. Field representatives will demonstrate the system to office personnel, follow up on any problems that arise, and keep in touch with new developments.

An exploration of superbill application in a medical practice office represents a small investment of time that could pay significant dividends in practice efficiency.

Superbill systems are produced by Control-o-fax, Safeguard, Caldwell and others, and are available through office supply houses.

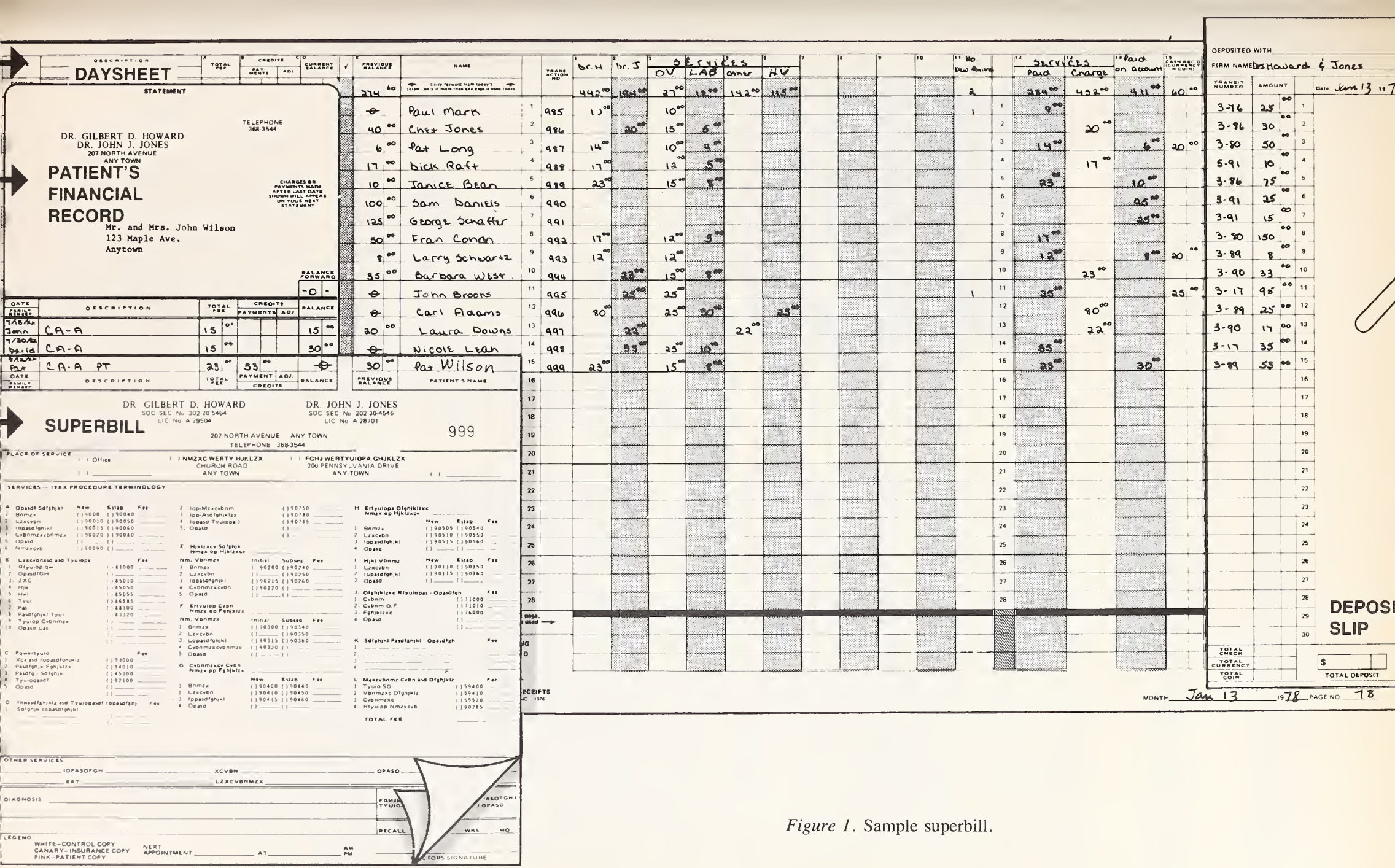


Figure 1. Sample superbill.

The President's Message



Dear Colleague:

A very important issue for the physicians of Kansas was presented at the recently completed House of Delegates meeting in Wichita. This issue, as revealed by Joseph C. Meek, Jr., M.D., Vice Chancellor of Academic Affairs, speaks to a shortage of clinical teaching material at the University of Kansas School of Medicine in Kansas City.

There are a number of valid reasons why this has happened. There has been a significant decrease in hospital admissions in many institutions throughout our state and nation. This trend has been spurred by rising costs, increased outpatient treatment, and perhaps is beginning to reflect some change in care because of lifestyle modifications. Another reason centers around the large number of well-trained physicians throughout the state who deliver a level of care that in past years was unavailable except at the Medical Center.

I would be remiss if I didn't mention the problem of poor communication during past years by UKSM staff with the referring physicians; this has also been a significant factor leading to a decrease in patient referrals. I would point out that this problem is not unique to our Medical School but is also faced by many leading institutions throughout the nation.

The important aspect of this situation is that the administration of the Medical School is committed to rectifying the communication gap and has pledged to do whatever is necessary to build a good referral base. A joint Medical Society-UKSM Committee has already met, and will continue to meet at regular intervals, to resolve any misunderstandings that pose obstacles to a solution.

Our negative experience of the past should not influence us in the future. We need a good, strong Medical School in our state, one that produces well-trained physicians. The School needs our help in providing patients from which students can develop sound basics of medical knowledge.

With the permission of the Council Districts, I plan to invite a representative from the Medical School to each of the meetings so that you will have an opportunity to share your thoughts and suggestions toward a solution.

I am committed to helping the Medical School with this problem. I ask you to join me in this endeavor.

President



Consultation Piece

The medical body has few blemishes that have not been exposed by its critics in recent years with implications of inherent corruption suggesting that the profession should not be — or not be allowed to be — long for this world. There are some deficiencies, however, which seem to have escaped them and these warrant occasional attention if only to assure ourselves we aren't changing too much — and even at the risk of providing those critics with new material.

For example, recurrent announcements from hospital records committees indicate that efforts to resolve that most durable of medical staff problems, getting those charts completed on time, has made little headway in the last forty years to our knowledge (and probably longer) despite electronic assistance, an astronomical increase in the number of record librarians, the long shadow of the JCAH, and threats to hang, draw, and quarter the delinquents. Human nature is a tough condition to overcome.

However, it is a different medical short-coming which has attracted our attention of late. Another persistent item of medical dysfunction is the sibling squabble that surfaces periodically between clinicians and the so-called ancillary services. The support team might be diagnosed as suffering from a professional "Rodney Dangerfield" syndrome since the pathognomonic feature is the certainty on the part of the "victims" that the clinician fails to accord the proper respect to his colleagues who devote their time to dispensing rays, looking through microscopes, or even holding the ether cone. The matter has been receiving some attention in the letters columns of the *JAMA* as a result of an article by a radiologist calling upon clinicians to consider such services equivalent to consultations and worthy of the same courtesies and regard that clinicians bestow upon each other in their consultative transactions (and goodness knows, *that's* a modest request considering the manner in which some of those are conducted). The fact that the issue has to be raised intermittently says something for its viability and provides for a little bilateral ventilation but bodes no good for the success of the current effort. Having worked both sides of the street, we claim a certain qualification for comment, something that can't always be said for these offerings.

The fact is, of course, that the clinician doesn't think of his requests for such services as bids for consultation with a true equal. He may consider these people as social peers, even, in the abstract, thinking of them as having some admirable features, but he reserves a special role for them — physicians but — well — *different*. Fundamentally, he considers his personal effort to be the one true professional expression and these service augmentations are, in this light, extensions of his own efforts and interests, having substance only by his mediation. And consciously or not, he presumes that whatever he knows or suspects about the patient is known to those who will function in these investigations — or at least enough that they can get the job done. He has graciously offered them the opportunity to come in from their detached worlds and conduct their special ceremonies and there is no need for them to fret themselves about the clinical details. And let us say, before we lose any clinicians still remaining in the audience, that this not-too-veiled charge of elitism is not so much a personal and positive effort of the clinician as an unfortunate by-product of the profession's growth. It may be that in this day of technologic dependence, it is a mechanism for retaining a certain integrity, a sort of denial that the service is on a par with his own efforts, however important its contributory need. Thus he diminishes any sense of relinquishment of his patient to the denizens of the optical-mechanical-electronic-vaporous world in which he may find himself a wandering soul as much

as the patient.

There was a time when the acme of such services was the blood count (superceding looking at the mucous membranes) and urinalysis (as the microscope demonstrated that there was more to it than holding it up to the light or tasting it). Then rays were discovered and the internal secrets of the chest and bony injuries were revealed. Today, these procedures are so commonplace that many offices are equipped to provide them and no loss of clinical status is felt. In fact, this availability is a convenience to the patient and, incidentally, a satisfying income producer. They are, in fact, called "routine," indicating that they have a basic necessity without necessity for any indication. Thus, the clinician is comfortable in the knowledge that he really could be doing other things if he weren't occupied with more important considerations, anchoring the concept that such services are just extensions of his own efforts and obscuring the thought that consultation philosophy might be desirable.

The office facilities have long since been unable to keep up with the unbelievable expansion of the ancillary horizons. At the outer limits, there are, indeed, areas of investigation in which the clinician has had to turn to the radiologist or pathologist or whatever just for orientation as to what studies to perform and why and what the results mean, so something like consultation enters in at this most sophisticated level. It is not surprising that the introduction of new procedures inevitably results in failure to use them at first when they could be useful, inappropriate or excessive use as they become better established, or inappropriate or incorrect interpretation of the results by the clinician, all of which could be corrected by a more informed exchange between the ancillary physician and the clinician. But the usual pattern persists in the majority of services: a bare order for the desired study is initiated. The name is usually given, sometimes the age and sex — rarely both. And let it be noted that the ancillary service often contributes at this point to the demeaning process since the average requisition slip is an abomination, designed for convenience rather than information sharing (except for that needed by the business office). There is usually a request for clinical information and the space allowed is roughly adequate for recording the chill index of South Yemen.

From the clinician's standpoint, consultation with a colleague of the conventional stripe is a time-honored sharing of responsibility, an exchange of professional wisdom, and a form of exemplary service to the patient. The ancillary service is of a different character. Whether the requisition is adequate or not, it is a "hired" service usually initiated by one of the office assistants — the clinician is too busy and anyway it is going to be handled by another assistant at the other end of the line — and the report when it returns, although signed by a fellow physician, is deemed to have been done by a technician, reinforcing the below-the-salt level of the whole transaction. Still, the effort emanates from the clinician's line of thought and he will be comfortable only with a strongly certain answer in its support. Even a contrary result is acceptable (although it does create some stress by requiring a redirection of thought) as long as it is offered with certainty. But let it not be indefinite — equivocation breeds contempt — for this testifies to the inepititude of the support team and hence all such services.

Despite ancillary egos and clinical myopia, there is of course an unassailable reason for bringing the communications to a mutual and equal (and consequently more effective) level of appreciation and respect: the benefit to the patient. The periodic introduction of the subject risks degeneration into an anecdotal harangue from each side which is the main reason the situation never changes very much. It comes down to the fact that the supporting service can provide more and better information regarding the patient if it has a more complete awareness of the problem. Many studies are explicit and perfunctory, and beyond the formal report there is little or nothing sought or to be given. But beyond these — and increasingly — a complete presentation of the patient's situation (comparable to that which would be supplied to a clinical consultant) can serve to direct the studies into more productive channels — even eliminate some when the intended purpose will not be served — or clarifying directions of investigation not previously appreciated. It is easy for the clinician to discount the radiologist's or pathologist's appreciation of the patient as a total clinical entity, presuming them to be concerned only with the technical aspects of their specialties, but if the clinical service has held the stage as the essential expression of medical function, the increasing technologic features of practice make it apparent that elimination of old concepts and relationships must be accomplished. We have yet to see the radiologist or pathologist who would not be glad to extend himself beyond the basic effort to exchange information and enhance the service if given the opportunity (some of them, in fact, would be hard to turn off).

It would be well, too, to resolve the conflict be-

(Continued on page 335)

KaMPAC Members

(Continued from page 283)

Richard J. Rupp, MD, R
Luecha Rutngamlug, MD, R
Michael E. Ryan, MD, R
W. Scott Ryan, MD, S
Mrs. W. Scott Ryan, S
Mrs. J. Alan Sanders, R
Peter C. Schram, MD, R
Alex Scott, MD, S
Steven G. Sebree, MD, R
Preston J. Shafer, MD, S
Joseph L. Shaw, MD, R
Robert N. Shears, MD, R
Lois E. Shortes, MD, R
Richard A. Siemens, MD, R
Robert E. Simmons, MD, R
Phillip B. Sisk, MD, S
R. M. Skibba, MD, R
Milo G. Sloo, MD, R
Boyd E. Smith, MD, R
Harold R. Smith, MD, R
Mrs. Harold R. Smith, R
Thomas W. Smith, MD, R
Arthur D. Snow, Jr., MD, R
Mrs. Marvin D. Snowbarger, R
Wayne E. Spencer, MD, R
Mrs. James R. Stark, R
Philip L. Stevens, MD, R
E. Kent Stevenson, MD, R
Karl W. Stock, MD, R
Morgan U. Stockwell, MD, R
Robert P. Stoffer, MD, R
G. Rex Stone, MD, R
Grant C. Stone, MD, R
Lawrence E. Stoskoph, MD, R
Chester H. Strehlow, MD, R
Charles M. Striebinger, MD, R
Henry B. Sullivan, Jr., MD, R
Tom G. Sullivan, MD, R
Sarohd Tangchupong, MD, R
Thomas L. Taylor, MD, R
Max E. Teare, MD, R
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Daniel M. Thompson, MD, R
Mrs. Daniel M. Thompson, R
Norman W. Thoms, MD, S
David E. Thurston, MD
Don R. Tillotson, MD, R
R. C. Tinker, MD, S
Mrs. R. C. Tinker, R
William B. Toalson, MD, R
John W. Travis, MD, S
Mrs. John W. Travis, S
Roger C. Trotter, MD, R
David L. Trudeau, MD
Virginia L. Tucker, MD, R
Wilson O. Uy, MD, R
John L. Vakas, MD, R
Carl A. Vierthaler, MD, R
Sunan Von Gkasemsiri, MD, R
Carroll D. Voorhees, MD, R
Theodore E. Wade, MD, R
William H. Walker, MD
Royce C. Walz, MD, R
Linda D. Warren, MD, C
Roger D. Warren, MD, C
Dale A. Waters, MD, R
Wallace N. Weber, MD, R
Kermit G. Wedel, MD, S
Mrs. Kermit G. Wedel, S
Lauren K. Welch, MD, R
Mrs. Lauren K. Welch, R
Maura S. Welch, MD, R
Claude J. Werth, MD, R
Darrell D. Werth, MD, R
Ren R. Whitaker, MD, R
Richard E. Whitehead, MD, R
Douglas M. Whitley, MD, R
Lowell W. Wilder, MD
Evan R. Williams, MD, R
Robert B. Wilson, MD, S
Mrs. Robert B. Wilson, S
Curtis V. Wolf, MD, R
Women's Clinic, P.A., S
A. J. Wray, MD, R
Mrs. A. J. Wray, R
Talaat E. Yaghmour, MD, R
Emerson D. Yoder, MD, R
John W. Young, MD, R
John O. Yulich, MD, S
Mrs. John O. Yulich, R
David L. Zacharias, MD, R
David S. Zamierowski, MD, R
Myron J. Zeller, MD, R

New Members This Month

Dale W. Anderson, MD, R
Ralph H. Baehr, MD, R
James L. Barber, MD, R
Franklin G. Bichlmeier, MD, S
Mrs. Franklin G. Bichlmeier, S
James W. Bruno, MD, R
Garland L. Campbell, MD, R
Richard C. Carleton, MD, R
Janet Chang, R
William J. Ciskey, MD, R
Tell B. Copening, MD, R
Peter J. Dalum, MD, R
George Dyck, MD
S. Jim Farha, MD, S
Norton L. Francis, MD, R
Mrs. Norton L. Francis, R
Jimmie A. Gleason, MD, S
Mrs. Richard M. Glover, R
Mrs. Phillip A. Godwin, R
Donald D. Goering, MD, R
Mrs. Donald D. Goering, R
Michael W. Good, MD, R
William N. Haffner, MD, R
Pratt Irby, MD, R
Kannampally J. Jacob, MD, R
Karl K. Kavel, MD, R
Theodore Lawwill, MD, R
Yong U. Lee, MD, R
Nancy Macy
Mrs. Robert F. Moore, S
Louis S. Morgan III, MD, R
Mrs. John Neuenschwander, R
Norman H. Overholser, MD, R
Daniel N. Pauls, MD, R
Carlos A. Prendes, MD, R
Norman K. Pullman, MD, R
Lew W. Purinton, MD, S
Zack A. Razek, MD, R
Charles S. Reeves, MD, S
Ivan E. Rhodes, MD, S
Daniel K. Roberts, MD, S
Mrs. James E. Roderick, R
Clarence E. Sherwood, Jr., MD, R
James M. Shields, Jr., MD, R
Newton C. Smith, MD, S
Jeffrey R. Steinbauer, MD, R
David E. Street, MD, S
Mrs. David E. Street, S
William K. Walker, MD, R
Mrs. William K. Walker, R

Official Proceedings

(Continued from page 280)

posefully than we have in the past. The Board, as you know, moves with glacial speed, and such promotional activities have been not so much rejected as simply not pursued. While ethics and propriety have ceased to be an issue, custom dies hard, but the Board — somewhat like the heirs who are interested in the demise of the principal because it will lead to the reading of the will — is not above putting past principles to rest if it means making a little money.

I do wish to say that, to the best of our knowledge, it has not been the burden of assuring our financial survival that has driven our former business manager to presumably greener pastures. Jerry Slaughter, as you know, has been a pleasure to have around and we trust that the umbilical cord that still connects him to the Kansas Medical Society will continue to nourish both ways. Meantime, Steve Carter has hardly had time to get his coat off before being subjected to the rhetorical marathon of the annual meeting. He should be somewhat prepared, however, since lawyers talk more than we do. So we welcome him and present him with a well seasoned staff which will be invaluable in his translocation from the judicial to the medical milieu.

In our own department, Val Braun and Eleanor Bell will continue their pattern of indispensability. Although the Board likes to promote its journalistic image, these women furnish the blood, sweat, and tears that go into the production of the *Journal*. Being the primary beneficiary of their good offices, I look forward to this annual opportunity to express to them my thanks for the times they have fronted for me, and the things they have taught me (although they may not have realized they did), and particularly the times they haven't been properly thanked before.

And now, since our President will be going home from here relieved of some heavy burdens, we wish to present him with seven and a half pounds of ballast in the form of bound *Journals* with the thanks and good wishes of the Board.

Constitutional Secretary —
Jack R. Cooper, M.D.

This is the Membership Report of The Kansas Medical Society for 1982:

Full Dues Paid Members	2,225
Associate Members	3
Delinquent Members	102
Emeritus Members	130

Golf Tournament Results
April 29, 1982

Championship Flight

1st Low Gross	John J. Schlueter, Wichita — Snyder Clinic Trophy
2nd Low Gross	James J. Muehlberger, Shawnee Mission
3rd Low Gross	Richard J. Cummings, Wichita
1st Low Net	Robert T. Manning, Wichita — Lattimore Traveling Trophy
2nd Low Net	Robert F. Moore, Caney
3rd Low Net	C. Leroy Williams, Wichita

First Flight

1st Low Gross	Herbert D. Doubek, Belleville
2nd Low Gross	David G. Laury, Ottawa
3rd Low Gross	Robert E. Riederer, Wichita
1st Low Net	David A. Leitch, Garnett
2nd Low Net	George F. Gsell, Wichita } tie
	Donald D. Goering, Coldwater } tie
Longest Drive	Robert E. Boyer, Kingman
Closest to Pin	Thomas F. Rosenberg, Wichita
Longest Putt	Thomas F. Rosenberg, Wichita

Honorary Members	1
Intern/Resident Members	37
LOA/Personal Exempt Members	24
Probationary Members	79
Retired Members	167
Service Members	15
Students	8
TOTAL	2,791

This compares with the membership in previous years as follows:

	Total Membership
1974	2,078
1975	2,137
1976	2,205
1977	2,267
1978	2,627
1979	2,876
1980	2,651
1981	2,751

Address by Isobel Dvorsky — President, AMA Auxiliary

Powerhouse Potential is a theme we use to describe our organization — the volunteer arm of the Medical Society. If you have not yet recognized the great potential of the volunteers of *your* Auxiliary — take a good look at them. Give them the opportunities, the resources, the direction, the push they need to work with you as a team. Once you bring the Auxiliary and Medical Society members together to solve problems or achieve goals and objectives, you will find that the Auxiliary will equal the enthusiasm, expertise, and effort of physicians — and that the job will be done more effectively and easily.

Auxiliary priorities are Medical Association priorities; our goals are your goals; only our methods differ. Our focus is community health education and services, legislative action, fund raising for medical students and schools, and support for the medical profession. Our purpose — to assist in improving health and quality of life for all people — is *your* purpose. Communication and cooperation are the keys that enable both Auxiliary and Medical Society to achieve their mutual goals.

Earl Thayer, Executive Director of the Wisconsin Medical Society, has called the Auxiliary "The single most unused asset of American Medicine."

We want to change that — with your help. Your Auxiliary can be your most viable, useful asset, for it is a strong volunteer organization with far-reaching expertise and ability. I urge you to establish close cooperation with your Auxiliary to achieve your mutual goals. Auxilians are truly your greatest advocates — and a powerhouse potential.

Address by Betty L. Moore — President, KMS Auxiliary

I am privileged and honored to be addressing this august gathering this morning. Robert, my husband, has decided that if this really is the last week of my job, he probably will keep me! It has been awhile since I have had time for home and housekeeping, but I knew that when I took this job — one I've enjoyed tremendously!

This year the Auxiliary has emphasized membership and AMAERF.

Membership is a constant area of work. Jan Meyer of the Missouri Auxiliary issued a membership challenge to us last fall; *i.e.*, that they would increase their membership more than we would by the end of this auxiliary year. Being a proud sunflower, what could I do? I accepted the challenge! I do not know how we stand at present, but I hope to be eating a nice big Missouri ham after the Chicago convention in June! This, of course, was all in fun, but with a serious purpose. We need members. They are vital to the life of an organization — and organization that is powerful and unique with 80,000 volunteers working for a common cause, namely safeguarding the nation's health. We hope you will be partners with us and encourage your spouse to join with us — we can do more together!

With the general decrease of federal funding, raising funds for medical schools is more important this year. AMAERF is our only fund raising project and is unique to us. No other organization that I know of cares about raising money to help with the education of those "rich" doctors. The Third Annual Auction is being held tonight with total proceeds going to AMAERF. We invite each of you to come, dig down deep, and have fun out-bidding your neighbor for that week of golf at Hilton Head, South Carolina, or that painting from Dr. Kernie Binyon — it's a beauty!

While these were our main concerns, we also continued the promotion of better health through our "Shape Up For Life" program with the emphasis on stress management this year. Dee Cauble (Wilbur G.), Wichita, continues with her caring and sharing for needy people around the world. This year alone she has sent 8,261 pounds of life supporting supplies to various areas around the globe.

Legislation is still of great interest to many of us as we actively participate in our "Legs Alert" system when a piece of less than desirable legislation is being written in our houses of government.

Our Learning Center on Body Pollution is still making the rounds of the schools to teach fourth and

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fifth grade children about the ill effects of drugs, poor nutrition, and poor mental health.

Last fall at the Auxiliary Confluence in Chicago, Dr. Sammons of the AMA won all hearts with his statement, "The greatest resource the AMA has is its Auxiliary." We certainly hope so, and will constantly strive to keep it that way!

So, now it is off with the old and on with the New. Meldon Laury (David), my good friend and traveling companion, will be installed as president tomorrow afternoon, and with her leadership, the programs promoting the health profession will continue and grow.

Thank you for your support of us and our efforts in assisting you in the creation of a better life through medicine.

KMS Ad Hoc Committee on Women in Organized Medicine — Joan Sehdev, M.D., Chairman

The Ad Hoc Committee on Women in Organized Medicine was conceived and appointed by Dr. Phillip Godwin, past president of the Kansas Medical Society, in June 1980. He set three goals for that committee to address: First, to identify Kansas women physicians who have leadership and other skills which would qualify them for committee and leadership roles; second, to increase active participation of women physicians at county and state levels; and third, to address the question of how to motivate women physicians to join the Kansas Medical Society.

It is of historical interest that in the same year, the American Medical Association also appointed a national committee to deal with problems of women in organized medicine. In December 1981, the AMA committee figures showed a national average for women physicians, both federal and non-federal, to be 12.0 per cent of physicians; for women residents, 22.0 per cent; and for women medical students, 27.5 per cent. This trend is also seen in the enrollment figures of the University of Kansas School of Medicine and the figures for active practice of medicine by women physicians in Kansas. In fact, the Kansas Medical Society statistics show that there were 139 women members in 1980, and this has risen to 170 women members in 1981. As of this presentation, two years after the inception of this committee, there are 15 women actively involved as members of committees for the Kansas Medical Society; two of these women chair committees.

It is of historical interest that Dr. Francena Porter, who joined the Kansas Medical Society in 1872, became the first woman member of a state medical

society. In addition, Kansas is one of only five states with a woman physician who is a member of the five-member American Medical Association Ad Hoc Committee on Women. This is Dr. Linda Warren, Hanover.

Of equal importance is the fact that Kansas is one of only five states with a committee whose purpose it is to seek out those women who are interested in more active participation in organized medicine.

We believe that women who practice medicine in Kansas represent a valuable potential resource for participation in organized medicine. In order to enumerate and utilize this resource, we sent a questionnaire to all women physicians who are members of the Kansas Medical Society. This basically consisted of a data base (qualifications, professional membership, previous participation in organized medicine, etc.); a question regarding interest in serving on a committee as a member or a chairman and if so, which committee; and a section soliciting comments regarding problems and thoughts concerning women's involvement in organized medicine.

Our response was unbelievable. We sent out 170 questionnaires to women members of the Kansas Medical Society. We received 100 responses. Tabulated, these indicated that 41 persons who responded were not interested, although some said they might be interested at a later time; ten who responded were retired and felt that they did not wish to participate; and 48 persons stated they were definitely interested in being a member of or chairing a committee for the Kansas Medical Society. If, from these 48, you subtract the 15 women who already are actively participating committee members or chairmen, that leaves 33 women who stated that they were very interested in participating on committees.

Responses indicated interest in most of the committees listed by the Kansas Medical Society, particularly Cost Awareness, Continuing Medical Education, the Impaired Physician Program, Legislative Committee, Maternal Health, and School Health.

A list of those women interested in being members of committees will be given to committee chairmen for their consideration. The list will include a short data base or resumé of each interested physi-

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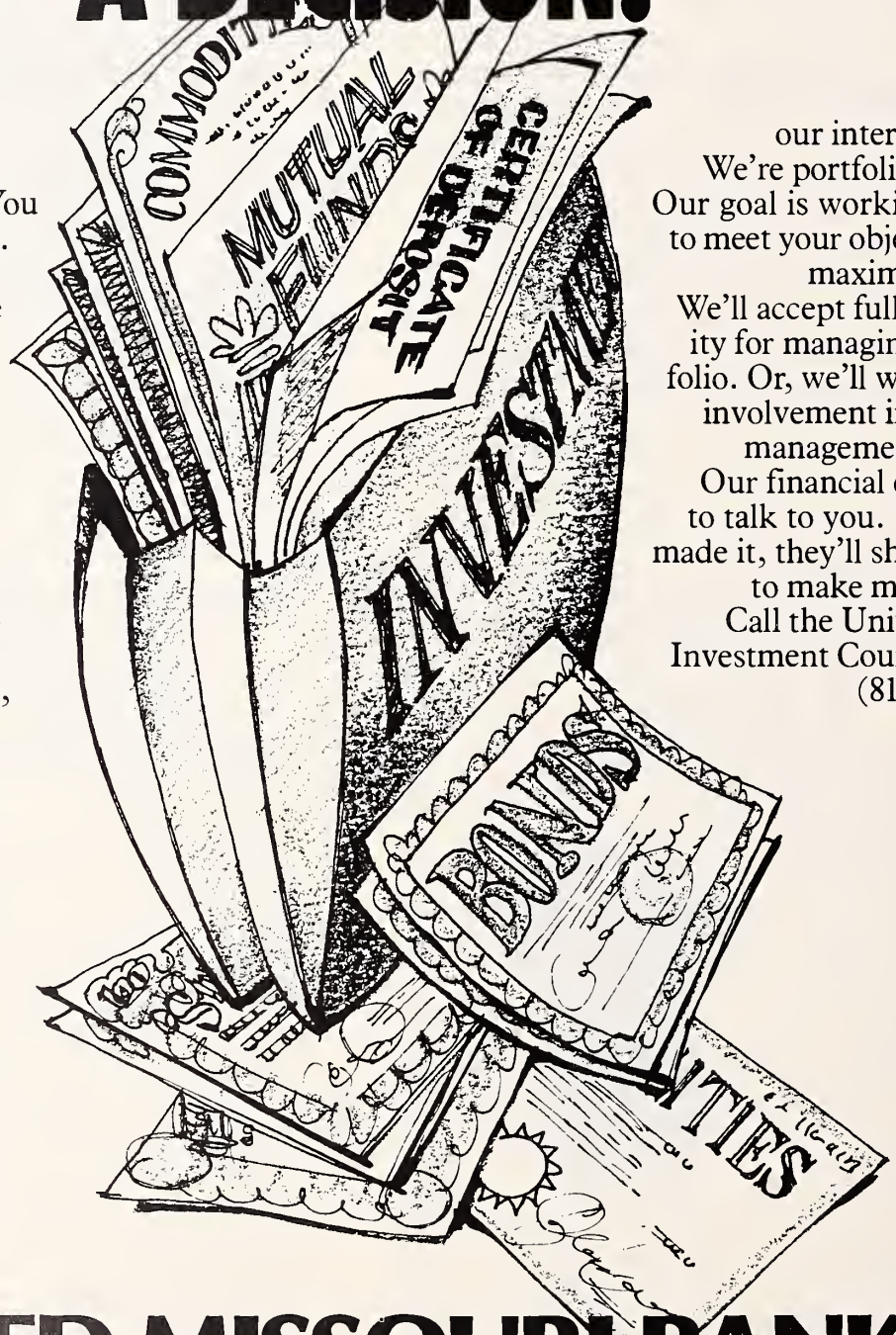
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cian which might assist the chairman in choosing qualified persons for his or her committee. A similar data base will be sent to the presidents of the respective local medical societies for their consideration.

I would like to stress that these physicians are interested in active participation because they feel that they can contribute to the committee if they are chosen to do so. They are not interested in token appointments as women members of committees.

It has been the aim of this committee to "mainstream" women by identifying them and bringing them into active participation in the Medical Society. That is not to separate them as a subgroup, but to integrate them as an active force based on individual participation and involvement at all levels of organized medicine — county, state, and national. It is important that this aim is the same as that of the national committee, and that this committee unequivocally states that it does not wish to remain as a continuing separate entity. Rather, it was created on an ad hoc basis to carry out the goals as enumerated, and when these goals have been achieved, the committee will no longer be needed.

There is one last goal remaining for this committee for the coming year — that is to deal with the question of how we can motivate women physicians to join the Kansas Medical Society. In addition, we would like to consider the question of what the KMS can offer to women in medicine.

Each year more women graduate as physicians. If organized medicine is to attract them as members, these women must be assured that a leadership role in the KMS is an achievable goal.

I would again like to express my sincere thanks and appreciation to Dr. Phillip Godwin for his keen interest and participation in this committee's activities. I would also like to thank our current president, Dr. Herman Hiesterman, for his support of the committee and its activities.

AMA Ad Hoc Committee on Women in Organized Medicine — Linda Warren, M.D., Member

I was appointed to this six member committee in June 1981 by the AMA Board of Trustees. The charge which we were given was two-fold: (1) to monitor the involvement of female physicians in organized medicine; and (2) to study ways to increase involvement in organized medicine by female physicians.

The first part of the charge is the one with the startling outcome — and it is here that we as physicians committed to organized medicine must pay attention or the future of organized medicine may be adversely affected. Approximately one in ten physicians today are female; in ten years, one in three will be female. Today only 26 per cent of female physicians belong to organized medicine as compared to 48-50 per cent of male physicians. The numbers speak for themselves: More women→less membership and involvement. We *must* change these trends if organized medicine is to continue to represent the American physician and to be effective in helping to form health care policies. This *is not* a feminist issue — it *is not* a struggle of male *vs* female — it *is* a struggle of organized medicine to see if it will survive in the changing demographic environment.

After analyzing these numbers, we have directed our attention to the second part of our charge. Among our activities have been: (1) working with the AMA Membership Department identifying target groups and areas where female membership is low; (2) including a special section on female recruitment in a membership kit to local and state societies; (3) preparing and sending lists of non-AMA members to local and state societies for recruitment; (4) doing mailings to female physicians nationwide; (5) achieving input into AMA Long Range Planning Council with emphasis on the importance of the female physician in the future of organized medicine; and (6) planning seminars by our committee as pilot projects in target areas, aimed at bringing organized medicine to the female physician at the *grass roots* level where the strategic link is located.

In December at the AMA interim meeting, Pam Formica, M.D., Chairman, and I met with the AMA Board of Trustees. Here we presented our analysis of the problem and recommendations with future plans. We feel we have the Board's support and encouragement in this problem and its solutions. The low rate of female membership and involvement is a potential threat to the future of organized medicine; however, a "threat" — once identified and analyzed — can become an "opportunity."

Kansas Medical Assistants Society — Mary Klinge, RRA, CMA-A, AAMA National Trustee

The Kansas Medical Assistants Society appreciates all of the help and support that the Kansas Medical Society has given us through the years. Members of this year's KMAS Advisory Committee are Drs. William R. Allen, Kansas City, Chairman; W. David Francisco, Kansas City; Warren E. Meyer, Wichita; Donald D. Moeller, Kansas City; Linda D. Warren, Hanover; and Wallace N. Weber, Hays.

I would like to ask those of you who have medical assistants who are not members of KMAS to encourage them to belong to their professional organization. Our primary objective is continuing education to enable us to serve our physician-employers more efficiently.

Our membership is tri-level — national, state, and local chapter. Kansas currently has 20 chapters throughout the state. These chapters meet on a monthly basis for continuing education, and some chapters have formed special study groups to prepare members for the national certification examination.

At the state level, we have at least two seminars each year. This past October one was held at Great Bend, and in March one was held at Wellington. Our State convention, which is convening this weekend in Topeka, has several educational sessions.

Our organization is not a union or collective bargaining agency, nor shall it ever become one. This is stated in our Bylaws at all three levels.

Because of inflation factors, our national dues were significantly increased this year. Our dues are $53 for state and national, and chapter dues range from $1-$3.

Again I would like to urge you to encourage your medical assistants to join their professional organization. Membership opens the door to benefits to aid the Medical Assistant's profesisonal growth and development.

KaMPAC —
Roger D. Warren, M.D., Chairman

KaMPAC is alive in 1982 in Wichita, but not quite well.

You, doctor, can help.

First, join KaMPAC — with your spouse — so that we can favorably impact the *very important* political process as it affects Kansas physicians.

The issues are numerous and vital. For example:

- Shall RNs practice medicine?
- How can we preserve and perhaps even improve medical school education in Kansas?
- Shall one unregulated group of medical practitioners — naturopaths — continue completely unregulated practice, perhaps to be joined by others?
- Cost containment. Accepted figures show that about 25 per cent of Medicare hospital expenditures are used by the provider, not in the provision of services, but in compliance (and documentation of compliance) with Medicare regulations. Another 15 per cent or more is used by the government in administration of the funds disbursed. We lose 40 per cent of our hospital Medicare dollar to the bureaucratic process! I remind you that entitlement to Medicare is essentially on the basis of age alone. It is obvious that huge amounts of money can be saved if government financed heatlh care is provided only for those who *cannot* pay for it themselves.

Through its KaMPAC members, KMS is strengthened so that our legislative committee and lobbyists can be more effective, both because of financial candidate support which you provide, and because of the constructive image projected by unified political action.

If you are one of the 400+ who have already joined KaMPAC, I thank you and assure you that your money will be wisely spent. If you have not done so, please write a check and give it to Gary Caruthers now. When you return home, tell your physician friends and their spouses to join.

Finally, whichever group you belong to — member or not yet member — drop by the KaMPAC Hospitality Suite this afternoon in the Cottonwood/ Pecan Room from 4:00 to 6:30 PM.

KaMPAC is alive in 1982 in Wichita, but not quite well.

You, doctor, can help.

REPRESENTING THE PROFESSION
Address by Robert D. Kelly, M.D.
AMA Trustee

It's a pleasure to join you here today and to share with you some of the many ways the AMA is representing our profession and the physicians of this state and in this hall in dealing with current problems and issues in medicine and in health care.

In a 1981 survey of physicians, both members and non-members told us that forceful representation should be our top priority. That's just where you'll find it — at the top of our priority list — and it *is* forceful.

The Courts

Often our representation takes place in the courts, as in our appeal of the U.S. District Court opinion that upheld the Federal Trade Commission in its advertising case. The four-four split decision by the U.S. Supreme Court in March had the effect of letting stand the lower court order affirming FTC authority over the professions. Although we are disappointed with the split decision, we see it as a clear signal that all the issues involved would be best addressed by the Congress, including the overriding issue of whether the FTC should even *have* jurisdiction over a non-profit voluntary association of professionals regulated by the state.

Therefore, the AMA intends to go to the Congress



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and continue to vigorously support two pieces of legislation already introduced: One bill would deny FTC jurisdiction over the professions; the effect of the second bill would be to impose a moratorium on any further extensions of existing FTC authority.

JCAH

Some contemporary issues and problems crop up within our medical-health care family and between our various organizations, and intramural problems require intramural solutions. So, at the direction of our House of Delegates, the AMA joined with other parent organizations in Joint Commission on Accreditation of Hospitals to address recurring concerns about how JCAH operates.

In responding to our concerns, JCAH has agreed to:

- Implementation of a three-year, rather than a two-year, accreditation cycle.
- A pilot test of the use of practicing physicians in addition to the Commission's own physician-surveyors.
- Development of a single set of standards for hospital-based psychiatric services.
- Pilot testing of revisions in the survey process to make it more responsive in terms of an action plan agreed to by the Joint Commission *and* by the hospital involved.
- Development of a more precisely targeted, tailored, and monitored accreditation cycle.

I think you will agree that *that's* representation — *forceful* representation — on your behalf.

The Legislative Arena

Your Association also continues to maintain real and present representation in Washington on your behalf; our new building there was dedicated yesterday. During 1981 alone, the AMA delivered 88 oral or written presentations in Washington on current issues and problems of concern to our profession, and made thousands of contacts with legislators.

We are also keeping a very close watch on pro-competition and consumer choice legislation now in the Congress, and on eventual health care implications of President Reagan's "New Federalism." AMA has publicly supported the concept of injecting more competition into the health care marketplace, so long as freedom of choice for physicians and patients is preserved and quality of patient care is not unduly compromised.

The AMA House of Delegates has adopted several principles to direct Association responses to competition-consumer choice legislation in Washington, including a possible bill by the Reagan administration — although they seem to have an on-again, off-again attitude toward such a bill. Secretary Schweiker spoke to us last night and didn't mention a competition bill.

The basic effect of the AMA's principles would be to change tax laws to enhance beneficial competition, thus encouraging cost-effectiveness in the health care marketplace. Such a policy would also widen the range of choices available to physicians and patients, with emphasis on less expensive insurance benefit packages — including deductibles and co-insurance — and on less expensive alternatives to hospitalization.

The AMA also has supported the concept of the President's block grant approach to federal funding of social entitlement programs, including health care programs, and certainly we support defederalization of the health care system. But, like you, we don't want to see funding of programs such as Medicare and Medicaid reduced to the point where quality of care — hence the patients themselves — will suffer.

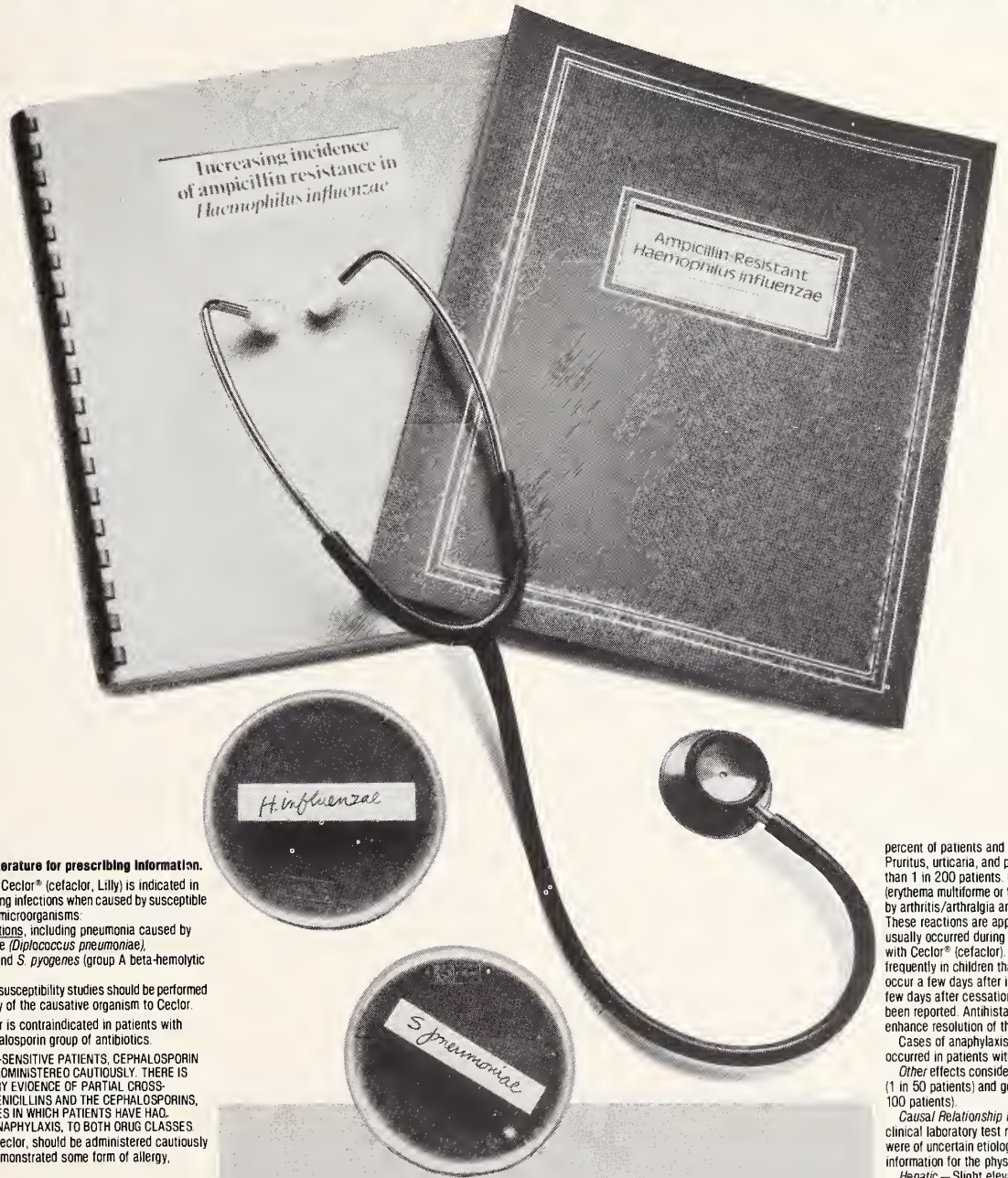
Rational Health Strategy

Until the President and the Congress reach legislative and budgetary agreement, precise impact of "New Federalism" will be unclear. One thing is clear, however — that tight economic constraints in this country, at both federal and state levels and in the general economy, are likely to continue for some time to come. We at the AMA have concluded that what the nation really needs are hard facts relating to how much the government can spend on those unable to help themselves and on how those funds can be spent in the most efficient and cost-effective ways.

This nation must get away from enacting or proposing patchwork fiscal fixes for patchwork social programs that the government took on during economic boom days of the 1960s. Therefore, the AMA itself is now undertaking a broad evaluation of health care policies that will assure citizens of needed care within existing economic constraints. We will soon be making a series of both short and long-term recommendations on how the nation might best resolve health care problems, including cost problems. Our longer-term recommendations will be based on an 18-24 month evaluation process that will involve many of the key players in the medical-health care community, including medical specialty societies, provider organizations, insurors, and others.

The AMA wants to take the lead in developing a national health strategy responsive to specific problems, and to outline appropriate roles for the public

An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor

Pulvules®, 250 and 500 mg

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci).

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coomb testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: Adverse effects considered related to cefaclor therapy are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70) and nausea and vomiting (1 in 90).

As with other broad-spectrum antibiotics, colitis, including rare instances of pseudomembranous colitis, has been reported in conjunction with therapy with Ceclor.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor® (cefaclor). Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [100281R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

1. Antimicrob. Agents Chemother., *8*:91, 1975.
2. Antimicrob. Agents Chemother., *11*:470, 1977.
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7. Data on file, Eli Lilly and Company.
8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

Lilly

Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

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and private sectors to play. We think that's the kind of leadership and representation we owe to America's physicians.

Political Action Committees

Freedom to express political views and to support candidates who share in those views has been an important part of America's democratic form of government. Last September, our American Medical Political Action Committee (AMPAC) held a political education conference in Washington on the occasion of its 20th birthday. During the course of those 20 years, AMPAC — and many state political action committees — have emerged as forceful representatives of what physicians believe is best in new medical and health legislation at federal and state levels.

In a communication read to the conference, Senator Wendell Ford (D-Kentucky) assessed just how forceful AMPAC had become: " . . . You people wrote the book on how to make a PAC work, and without question your organization's record of success stands alone."

Edward Rollins, Deputy White House Assistant for Political Affairs, said part of the success of the Reagan Administration " . . . has been due to the efforts of groups such as AMPAC."

There are hard statistics to prove Senator Ford and Mr. Rollins knew whereof they spoke. In the 1980 national elections, AMPAC supported — on a bipartisan basis — 426 candidates for the House and Senate. About 81 per cent, or 326 candidates, were subsequently elected.

The recent AMA survey revealed that 65 per cent of physicians polled believed that as citizens as well as physicians, our profession has a responsibility to contribute to worthwhile political causes, and thus provide the kind of political representation that AMPAC has been and is giving you. Unfortunately, the same opinion poll uncovered a disturbingly large number of physicians who had seldom if ever been asked to join or contribute to medical PACs.

In these troubled times, when we often feel disenfranchised as individuals, medical PACs and our federation offer individual physicians an opportunity to participate in and contribute to our democratic form of government. The fact that we are citizens as well as professionals, and that we have attendant responsibilities, is not a new concept.

It is notable that in 1981, KaMPAC ranked second nationally in contribution per member, and for the fifth consecutive year has increased its membership.

The Coalition Movement

A promising example of what voluntary, private sector free enterprise can accomplish is the rapid growth of local health care coalitions in communities. An outgrowth of a series of 1978-79 meetings between AMA officials and executives from nearly 100 of America's largest corporations, the rapid growth of these coalitions has been fostered by cooperation and creativity of hundreds of local medical society, business, and civic leaders. Although the movement is only about 2½ years old, about 50 of these coalitions already have been established, with many more in the planning stage.

We at the AMA believe that the coalition movement is especially promising for the future precisely because responsibility for cost restraint is put right back where it belongs — in the states, counties, and local communities.

That's where the problems are, and that's where the solutions are.

To add even more momentum to the movement, in January a national coalition was created by representatives of the AMA, the American Hospital Association, Blue Cross-Blue Shield, the Health Insurance Association of America, the Business Roundtable, and the AFL-CIO.

That's the kind of representation — *interfederation* representation — that we sorely need to enhance the socioeconomic and political *visibility*, and hence the socio-economic and political *credibility* of American medicine with the American people.

Communications, Publications

The AMA survey of member and non-member physicians also revealed a belief that the AMA should continue to provide up-to-date scientific and medical-health related information to the profession and to the public. Within the past few years we have become the largest compiler and communicator of such information in the world. Our annual publication numbers include:

- 14 million copies of *American Medical News*.
- 14 million copies of the English-language edition of *JAMA*.
- 250,000 copies of nine medical specialty journals each month.

This is a total for 1981 periodicals of 32 million copies. *JAMA* also is now being published in several foreign language editions for circulation in other countries, including Japan, France, Belgium, Switzerland, West Germany, and the People's Republic of China.

A focal point for the development of definitive

scientific reports is the AMA's Council on Scientific Affairs. Last year this council prepared, and AMA Delegates adopted, reports on such topics as:

- Risks of nuclear energy and low-level ionizing radiation.
- Drug abuse related to prescribing practices.
- Organ donor recruitment.
- Electronic fetal monitoring.
- Improving health care for homosexuals.
- "Agent Orange" health effects and dioxin contaminants.
- Genetic counseling and prevention of birth defects.
- The prescription of tranquilizers and antidepressants for women.

In addition, we send out thousands of newspaper, radio, and TV positive personal health promotion tips each year. These reach millions of American readers, listeners, and viewers.

A logical extension of the AMA's comprehensive communications role is our work with General Telephone and Electronics in developing a "first-of-its-kind" computerized National Medical and Health Information Network. The network, which will begin operation late this year, will be knowledge-based rather than bibliographic in nature, and eventually will disseminate up-to-the-minute clinical information and protocols to computer terminals in physicians' offices in all sections of the country. The first phase of the network will incorporate drug knowledge and drug therapy and reaction information from essentials of an AMA book, *Drug Evaluations*.

The national computer system also will serve other medical users, such as medical schools and hospitals, as well as other health care institutions, to address administrative, socioeconomic and political concerns related to medicine and health care.

As communicator and publisher, then, the AMA also is a forceful and authoritative representative for American medicine nationally and internationally.

Summary

This is just a part of the story, the true and powerful story, of how the American Medical Association is working hard to forcefully represent America's physicians. It's a story that helps explain why the franchise in our House of Delegates has expanded to 304, and now represents virtually all of American medicine. It's a story that helps explain why membership in all categories was up last year, and why our student and resident memberships have grown to about 24,000 within just a few years.

Given the medical and health care challenges and opportunities that lie ahead of us, and the need for even stronger membership — hence stronger representation for our profession — it's a story that must be told, far and wide. We have a great story to tell — both to our profession and to the public. We're asking *you* to help us tell it.

The Kansas Foundation for Medical Care, Inc. — Louis M. Culp, M.D., President

Status Report

- PSRO review was implemented in three psychiatric specialty hospitals in addition to the 145 acute care hospitals in Kansas.
- Ancillary services review was implemented January 1, 1982.
- A proposal similar to Sedgwick County's Foundation for Medical Care Plan has been submitted to SRS for review of Medicaid patients. The proposal includes review in an eight-county area, plus continued delegated review in the rest of the state. SRS is requesting admission certification in the first 24 hours and non-delegated review in the eight-county area. Preadmission certification for non-emergency admission is being considered by SRS. July 1, 1982, is the proposed starting date for KFMC's review plan.
- KFMC's modified review plan has been expanded. This new system, known as data based review, is a retrospective review of data to determine where to do concurrent review. *Retroactive denial is not a part of this process*. Overall, this should adequately focus in on problem areas and be more economical.
- The common or unique physician identification number has stirred considerable response. As of two years ago, it became apparent that unique identification numbers were needed. At that time, Nader's organization was suing the PSRO in Washington, DC, for release of data. Through the efforts of the PSRO and the AAPSRO, PSROs were declared, in the Court of Appeals this winter, not to be a government agency, and therefore not subject to the Freedom of Information Act. Mr. Nader did not appeal to the Supreme Court. This is as good or better protection than legislation. Your KMS, State Board of Healing Arts, Medicaid, The Blues, the fiscal intermediaries have a common number. Why shouldn't we? We are your physician-run Peer Review Organization established with your blessing, endorsed by 1,990 members and growing — greater than 65 per cent of the physicians in Kansas. Apparently concern about confidentiality is the trouble maker. We pledged confidentiality from outside organizations

Famous Pairs.

They work so well together.

One of man's most amazing explorations and scientific adventures, the successful Gemini flight program was a triumph of imagination and—teamwork. Two men learned to operate in space, to rendezvous, to dock, and to work outside their spacecraft in the hard vacuum of outer space. Not only did they coordinate their efforts with ground backup, they also complemented each other's activities within the close confines of the space capsule.



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Anusol-HC® & Tucks®

...another well-known pair that works so well together! Ninety-five percent of colon/rectal surgeons surveyed added Tucks pads concomitantly to hemorrhoidal treatment programs they recommended.*



Anusol-HC®

Suppositories/Cream with Hydrocortisone Acetate

*The #1 physician-prescribed product for hemorrhoids and other common anorectal disorders***

- ☐ *Antiinflammatory, to relieve edema, burning, itching, pain*
- ☐ *Astringent, to help promote healing*
- ☐ *Emollient, for easier bowel movements and soothing relief of local trauma*

And, when pain is a special problem, Anusol Ointment offers the benefits of the anesthetic, pramoxine HCl.

TUCKS®

Pre-Moistened Hemorrhoidal/Vaginal Pads

The #1 hemorrhoidal pad† for added external relief and gentle cleansing of fecal residue

- ☐ *Soothes, cools, comforts the irritation and itch of hemorrhoids and other common anorectal disorders*
- ☐ *Hygienic rectal wipe—an integral part of the anorectal regimen*

Once pain and inflammation subside, for dual action recommend regular ANUSOL®—to maintain patient comfort—and TUCKS®—to maintain patient anorectal hygiene.

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WARNER LAMBERT

*Meeting of Am Soc Colon/Rectal Surgeons, May 1980.

**Based on total prescriptions filled for hemorrhoidal preparations during the first three quarters of 1981. The National Prescription Audit, IMS America Ltd, Sept 1981.

†1981 data from leading marketing research organization.

PD-85-JA-0867-P-1 (2-82)

ANUSOL-HC® Suppositories/ ANUSOL-HC® Cream

Before prescribing, please see full prescribing information. A Brief Summary follows:

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in external and internal hemorrhoids, proctitis, papillitis, cryptitis, and fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

CONTRAINDICATIONS

Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

WARNINGS

The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

PRECAUTIONS

General

Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

DOSAGE AND ADMINISTRATION

Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

Store between 59°-86°F (15°-30°C)

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and until confidentiality was established and protected, we did not want to know any form of identification. Now, we and you should be protected. There are other ways of accomplishing this unique common identification number, but the present requests for your numbers is the simplest approach.

- The quality assurance program has been revised and implemented. There still are some bugs to be worked out and clarifications to be made. It is certainly not the intent of KFMC to duplicate or create medico-legal problems. Some duplication with respect to JCAH is necessary since KFMC by its very nature and purpose must monitor the quality assurance program.
- KFMC has formed a division called PRK — Peer Review of Kansas — with Richard Brownrigg, M.D., Dodge City, Chairman. The purpose of PRK is to enter into private review, such as with individual companies, labor groups, consumer organizations, and insurance companies. We are in the process of contracting in the Kansas City Metropolitan Area. This should be operational this fall. We are in the process of contracting in the Kansas City Metropolitan Area. This would be operational this fall. We will do review on the Kansas side and Northwest Missouri PSRO on the Missouri side. We are in the process of negotiating with other groups.
- Two of our Board members and one of our staff now serve on the National American Association of PSRO Committees: Larry Pitman, Review Services Committee; Dean Collins, M.D., Long Term Care Committee; and Louis Culp, M.D., Legislative Affairs Committee.
- Our Program and Development Committee is gearing up under Erwin Janssen, M.D., Chairman, to explore other avenues of review, such as long term care. Other services considered in the future are in data services, medical abstract systems, and administrative services such as fiscal agents.

Legislation

Two bills have been introduced in the Senate by Senators Durenberger and Baucus — SB 2142 and SB 2150. They are also supported by Senators Heintz, Moynihan, and Thurmond. Senator Durenberger states his bill is to "redirect, simplify, and enhance the cost effectiveness of the PSRO program under Medicare." Further, he states, "I'm convinced that we cannot abandon the program nor can we afford to retain it in its present form." Generally, the bills strengthen the resulting review organizations. The KFMC, the AAPSRO, and its Legislative Affairs Committee oppose the bills' position on giving equal eligibility to be the review organization to fiscal intermediaries or to for-profit insurance companies. The National Association of Health Insurance, HIAA, also opposes this. The bill does emphasize that the review organization should be a statewide organization. Sanction recommendations are strengthened. Private review is encouraged. Review organizations would not be considered a federal agency and would not be subject to the Freedom of Information Act. Not included in the bill, but stated in the testimony, was the plan to make all review non-delegated. The KFMC and the AAPSRO oppose this. At the hearing of the Senate Finance Committee's Subcommittee on Health, April 1, 1982, there were numerous testimonies for the bills with a few changes. Three groups testified against the proposed bills: The Administration, the AMA, and Congressman Ron Paul of Texas, who also advocated the repeal of Medicare and Medicaid. It's not legislation, but there is a pilot study being done for hospital reimbursement on a diagnosis basis entirely, and a recommendation by J. Alexander McMahon, President of the American Hospital Association, that reimbursement be done on a capitation basis only. If such should occur, both cost effective and quality control review will become more important and necessary.

Budget Cut

The Administration's budget cuts have caught up with us at the beginning of our fiscal year — April 1, 1982. The highways and telephones were kept hot between Topeka and Kansas City, and between Topeka and HCFA in Baltimore for two weeks. Shifts in personnel between parts of the budget were tried; some were allowed, some were denied. Finally, after a very difficult and very emotional Executive Committee meeting, Jim Agin was released as Executive Director of KFMC on April 9, 1982, and Larry Pitman was made Executive Director on April 12, 1982. The decision was made entirely on the priority of keeping the Foundation viable as your peer review organization.

The Executive Committee of the Foundation will be submitting a resolution in honor of Jim later today. We ask for your support.

Election Results

President — Louis M. Culp, M.D., Kansas City; Vice President — Richard Glover, M.D., Newton; Secretary — G. Rex Stone, M.D., Manhattan; Treasurer — George R. Learned, M.D., Lawrence.

New members of the Board are:

Two-year slotted — Gary A. Kerns, M.D., Fort Scott



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CPAs. The initials tell you their credentials. The Yellow Pages tell you their names.

Two-year non-slotted — Donald Moehler, M.D., Kansas City; Katherine Pennington, M.D., Wichita; Erwin Janssen, M.D., Topeka; Michael McGinnis, M.D., Dodge City.

One year slotted — Allen D. Grillis, D.O., Coffeyville

One year non-slotted — Richard Gruendel, M.D., Kansas City; Alex Scott, M.D., Junction City; Robert W. Brown, M.D., Salina.

Summary

It appears that accountability and quality control by review by some organization are here to stay. We, as members of KFMC, are concerned that this be a physician peer review organization, not a fiscal intermediary.

SECOND SESSION

The Second Session of the House of Delegates was called to order by the Speaker, Clair C. Conard, M.D., on Sunday, May 2, 1982, at 9:00 AM at the Hilton Inn East, Wichita.

The Speaker placed some rules before the body and announced that *Sturgis Standard Code of Parliamentary Procedure* would be followed at this meeting. Every Delegate will be given an opportunity to be heard on every question, but except for the person who makes the motion, all Delegates were asked to cooperate by being heard only once upon a single question. He also announced that a quorum was present.

The Speaker appointed the following tellers: Drs. Franklin G. Bichlmeier, Kansas City; Herbert Fransen, Newton; and David A. Leitch, Garnett.

Emerson D. Yoder, M.D., President of MEDISERVE, Inc., was invited to give a report. Dr. Yoder briefly reviewed the history and the goals of that organization and stated that 67 students are presently in the MEDISERVE program, 13 of whom will graduate this year. Because of the existence of the state scholarship program for medical students, the Board will not be issuing new loans. Since 1977, the Kansas Medical Society and the Kansas Farm Bureau have each contributed $64,000 to this project (of which $24,000 was a loan). It appears that an additional $12,500 will be required from each contributor by December 1982.

The following election results were reported:

President Elect: Jimmie A. Gleason, M.D., Topeka

First Vice President: F. Calvin Bigler, M.D., Garden City

Second Vice President: Clair C. Conard, M.D., Dodge City

Constitutional Secretary: K. William Bruner, Jr., M.D., Wichita

Treasurer: William K. Walker, M.D., Sedan

AMA Delegate: Alex Scott, M.D., Junction City

AMA Alternate Delegate: Warren E. Meyer, M.D., Wichita

The following Council district elections were reported:

District 2: Louis M. Culp, M.D., Kansas City

District 4: George W. Pogson, M.D., Pittsburg

District 11: K. William Bruner, Jr., M.D., Wichita

District 13: Wallace N. Weber, M.D., Hays

District 14: Donald E. Beahm, M.D., Great Bend

District 15: Edwin D. Rathbun, M.D., Liberal

District 19: Robert J. Haskins, Chanute

Dr. Hiesterman advised the House that the KMS-UKMC Liaison Committee will be reactivated immediately. This decision was the result of a meeting held yesterday by the representatives from both organizations.

Richard A. Guthrie, M.D., Chairman of the Scientific Program Committee, expressed his gratitude to the members of his planning committee for the excellent program presented. He also acknowledged the considerable administrative assistance provided by Mr. Gary Caruthers and Ms Diane Henderson.

Kermit G. Wedel, M.D., the newly installed President of the Kansas Medical Society, addressed the House and stated that among the projects for the coming year will be the issues of cognitive review, public health, University of Kansas Medical Center, as well as the involvement of the various KMS standing and ad hoc committees. He invited input from members of the Society for suggestions and comments. Dr. Wedel thanked the House for the opportunity to serve as its President and looked forward to the coming year.

Dr. Clair C. Conard, Speaker, expressed his appreciation to the Reference Committee for the excellent work in preparing the report. He also thanked the Medical Society of Sedgwick County for hosting an excellent meeting this year. Dr. Conard thanked Dr. Robert Kelly for taking the time to attend the entire meeting and to address the House of Delegates.

The Speaker announced that future annual meetings have been scheduled as follows:

1983	May 5-8	Downtown Ramada Inn, Topeka
1984	May 3-6	Holidome, Hutchinson
1985	May 2-5	Broadmoor Hotel, Colorado Springs

G. Rex Stone, M.D., Manhattan, was then installed as the new Speaker of the House of Delegates, with Franklin G. Bichlmeier, M.D., Kansas City, serving as Vice Speaker. The House gave a standing ovation to Dr. Conard for his excellent and outstanding service as Speaker of the House of Delegates for the past 12 years.

There being no further business, the meeting adjourned at 11:25 AM.

An asterisk following the resolution number indicates a change in the Constitution and Bylaws.

RESOLUTION NO. 82-1

Forum for Health Care

WHEREAS, The country is in a period of review and reassessment of private and public policies relating to the costs, planning, and delivery of health care; and

WHEREAS, The AMA strongly supports health care forums that include meaningful physician participation, so that primary emphasis is given to quality medical care, including availability and access, as well as recognizing the importance of cost effectiveness and cost containment; therefore be it

Resolved, That the Executive Committee of the KMS take the lead in calling together interested organizations to explore the possibility of organizing a health care forum for Kansas.

RESOLUTION NO. 82-2*

Consent Calendar

Resolved, That in Section 5.4413 all wording after the word "number" be deleted and a period (.) be inserted after "number"; and be it further

Resolved, That a new Section 5.44211 be adopted stating the following:

> If, in the opinion of the reference committee chairman, a resolution can be acted upon without additional discussion, he shall direct the resolution to be placed on the consent calendar. A list of all such resolutions shall be presented at the end of the reference committee report. The Speaker shall ask if there is any objection. If an objection appears to any resolution, it shall be removed from the consent calendar and placed before the House for individual consideration. If no objection appears, the resolutions on the consent calendar will be placed as a package before the House for action.

RESOLUTION NO. 82-3

Medical Examiner System

WHEREAS, Crime is rated as one of the most important problems facing our society; and

WHEREAS, The scientific determination of the cause and manner of death has become an increasingly more important aspect of deaths falling within the realm of public interest; and

WHEREAS, A survey of Kansas coroners indicates the need for a statewide medical examiner system; therefore be it

Resolved, That the Kansas Medical Society study the concept of a statewide medical examiner system; and be it further

Resolved, That the Kansas Medical Society encourage the State Legislature to establish an interim committee to study the need and manner of establishment of a statewide medical examiner system.

RESOLUTION NO. 82-4

Membership Application Procedures

(Referred for Study)

WHEREAS, More physician members are needed at the state and county level; and

WHEREAS, Application procedures at the state and county levels are tedious and repetitious; and

WHEREAS, These application procedures could be barriers to membership; and

WHEREAS, The Kansas Medical Society should encourage simplification of membership application at the state and county level; therefore be it

Resolved, That the Kansas Medical Society appoint a committee to coordinate and streamline state and county membership application.

RESOLUTION NO. 82-5

Reduced Dues Provision — First and Second Year of Practice

Not adopted.

RESOLUTION NO. 82-6*

Members (1st Year) Dues Reduction

Resolved, That the By-laws be amended as follows:

1-6111: Active members (1st year) — Physicians in their first year of full time medical practice after completing their residency requirements. They shall pay *50 per cent* of regular dues and assessments the first full dues year.

RESOLUTION NO. 82-7*

Federal and State Full-Time Employment Membership Category

Not adopted.

RESOLUTION NO. 82-8*

Military Membership Category

Not adopted.

RESOLUTION NO. 82-9*

Reestablishment of Medical Student Society

WHEREAS, Resolution 81-13 was adopted with the intent to increase medical student membership; therefore be it

Resolved, That the By-laws be amended to provide that student members shall receive *The Journal* free of charge.

RESOLUTION NO. 82-10

Organ Donation Education Program

WHEREAS, Medical and surgical technology of recent times has made it possible to transfer an organ of one individual to another; and

WHEREAS, Organ transplantation has the potential to greatly enhance the quality and quantity of life for the recipient; therefore be it

Resolved, That the Kansas Medical Society support a program making the general population aware of organ donations and the appropriate procedures to follow if a person would like to donate an organ or organs for transplantation.

RESOLUTION NO. 82-11

Guidelines for Physicians Attending Patients in Long Term Care Facilities

WHEREAS, The Long Term Care Committee is interested in improving nursing home care and encouraging more physician involvement in nursing home care; therefore be it

Resolved, That the Kansas Medical Society endorse the AMA Guidelines for Physicians Attending Patients in Long Term Care Facilities; and be it further

Resolved, That copies of the Guidelines be distributed to all members of the Kansas Medical Society.

RESOLUTION NO. 82-12

Appointments to the Kansas State Board of Healing Arts

Not adopted.

RESOLUTION NO. 82-13

Commendation for Jerry Slaughter

WHEREAS, The Kansas Medical Society has enjoyed the services of Jerry Slaughter as Executive Director since 1975; and

WHEREAS, Jerry has exhibited outstanding intellect, deportment, and eloquence on behalf of the Medical Society; and

WHEREAS, Jerry has won many friends for the medical society by his friendly and open manner; and

WHEREAS, The physicians of Kansas have benefited by and appreciate the efforts put forth by Jerry, and will miss Jerry now that he is embarking on new endeavors; therefore be it

Resolved, That the Kansas Medical Society take this opportunity to thank Jerry Slaughter for his "Job well done" since 1975 and to wish him well in his new endeavor.

RESOLUTION NO. 82-14

Kansas State Board of Healing Arts — Legal Counsel

WHEREAS, The Kansas Medical Society fully supports the role and function of the Kansas State Board of Healing Arts; and

WHEREAS, Legal counsel is basic to the day to day activities of the Board; and

WHEREAS, Current state law provides that the State Attorney General will assign from that office's staff of attorneys legal counsel to the Board; and

(Continued on page 334)

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J-9043-4 July 1981

The Physician's Sleep Glossary

Some common sleep laboratory terms

poly•som•no•graph. An instrument which simultaneously records by electrodes physiological variables during sleep—for example, brain activity (EEG), eye movements (EOG), muscle tonus (EMG) and other electrophysiological variables. These readings indicate precisely when patients fall asleep, how many wake periods they experience, the quality of sleep and the duration of sleep.

sleep la•ten•cy. The period of time measured from "lights out," or bedtime, to the commencement or onset of sleep.

wake time af•ter sleep on•set. Intervals of time spent awake between onset of sleep and the end of the sleep period. The polysomnograph registers the length and frequency of the intervals.

to•tal sleep time. The amount of time actually spent in sleeping. This is estimated by subtracting wake times from the period encompassed by the onset and the termination of sleep.[1]

REM/NREM. 1. REM, or rapid eye movement, sleep is "active"—characterized by increased metabolic rates, elevated temperature and arousal-type EEG patterns. 2. NREM, or non-rapid eye movement, sleep represents "quiet" sleep stages. There are four distinct stages of NREM sleep.[2]

re•bound in•som•nia. A statistically significant worsening of sleep compared to baseline on the nights immediately following discontinuation of sleep medication.[3]

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More total sleep time on nights 12 to 14 of therapy[4] and continued efficacy for up to 28 nights[5] with

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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Diabetes II

(Continued from page 294)

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Resolutions

(Continued from page 332)

WHEREAS, It is the Society's opinion that in order to function at maximum effectiveness, the Board should have the authority to employ and discharge its legal counsel; therefore be it

Resolved, That the Council of the Kansas Medical Society review this matter and initiate the appropriate action which will allow the Kansas State Board of Healing Arts to employ their own legal counsel.

RESOLUTION NO. 82-15

Dues for Resident Members

Not adopted.

RESOLUTION NO. 82-16

Membership — Kansas Blue Cross Board of Directors

WHEREAS, The executive committee of Blue Cross at its March 11, 1982, meeting recommended that the Blue Cross bylaws be amended whereby only physicians who participate with Blue Shield may serve on the Blue Cross Board; and

WHEREAS, Until this proposed amendment is acted on by the full Blue Cross Board of Directors at their May 6, 1982, meeting, non-participating physicians are permitted to serve on the Blue Cross Board of Directors; and

WHEREAS, All licensed Kansas physicians and surgeons who provide professional services to Blue Cross/Blue Shield subscribers should have the opportunity to serve on this board; therefore be it

Resolved, That the Kansas Medical Society voice its opposition to the proposed Blue Cross bylaw amendment which would limit physician membership on the Blue Cross Board of Directors to participating physicians only.

RESOLUTION NO. 82-17

KMS Trust Fund For Impaired Physicians

WHEREAS, The Council instructed the Impaired Physician Committee to develop a plan to assist the needy physician and/or their families; therefore be it

Resolved, That the following recommendations be adopted:

The purpose of the Kansas Medical Society Impaired Physician Trust is to provide a revolving resource for interim loans or occasional bequest to member physicians who are destitute or impaired and who are in dire financial straits due to illness. The administration of the Trust Fund will be as follows:

1. Because direct operation of a loan or benevolent fund would endanger the tax exempt status of the Kansas Medical Society, a separate entity (trust or non-profit corporation, as deemed equitable by KMS Legal Counsel) will be established on January 1, 1983.
2. The Kansas Medical Society will provide a start-up loan of $15,000 to maintain the fund until additional revenues can be raised.
3. Funding for the Trust Fund will be on a voluntary basis, through billing included in the annual KMS dues statements to members for contributions of $5 per year for five (5) years (the first billing to be in 1982 for 1983 contribution). Additional funds will be solicited from individual KMS members as voluntary tax deductible contributions; from KMS Auxiliary local chapters, and by other appropriate means.
4. Eligibility for loans or grants will be limited to KMS membership, either past or present, who are residents of Kansas.
5. Loans to needy impaired physicians will be made in amounts up to $5,000 per case, with interest charged at the level of the current prime rate. The committee will have some discretion as to interest payment and rate in special cases. Loans may be made directly to the individual or used to purchase term life insurance; tied into the "UKSM Retread Program," if such is established; or as individual circumstances may warrant.
6. Grants can be made to a physician's family depending upon individual circumstances and need. A grant will be limited to $2,000 and will be non-interest bearing and repayable at the discretion of the IPP Committee.
7. Requests for loans and grants will be administered by the IPP Committee in the following manner: The request will be referred to the Committee member geographically nearest to the applicant for investigation; the results of the investigation will be forwarded to the Trust Fund Subcommittee who will either approve or disapprove the request.
8. Funds will be available for the investigating physician to obtain services of a social worker in the area for the purpose of evaluating and reporting the situation of a physician or family making the request for grant or loan

and be it further

Resolved, That this resolution supercedes Resolution 81-1.

RESOLUTION 82-18

Nursing Home Aides

Resolved, That the Kansas Medical Society

oppose a change in regulations which would allow medication aides to give injections in nursing homes; and be it further

Resolved, That the Kansas Medical Society oppose the use of untrained nursing home aides without nursing or medical supervision.

RESOLUTION 82-19

Physician Advertising

WHEREAS, The recent decision by the United States Supreme Court appears to have settled the question on the standards that may be applied by states when regulating advertising by professionals; therefore be it

Resolved, That the Legislative Committee be directed to work with the Kansas State Board of Healing Arts to develop appropriate amendments to the Healing Arts Act which establish the standards of only prohibiting advertising that is false, fraudulent, misleading, or deceptive.

RESOLUTION NO. 82-20

Financing For Local Health Departments

(Referred for study)

WHEREAS, In recent years there have been several attempts to increase the amount of state funding for programs in local health departments; therefore be it

Resolved, That the Kansas Medical Society support the concept of increased state funding for local health departments.

RESOLUTION NO. 82-21

Regional Poison Center

Resolved, That the Kansas Medical Society support the establishment of a state-funded regional poison control center.

RESOLUTION NO. 82-22

Commendation for James E. Agin

WHEREAS, James E. Agin has faithfully served for eight years as Executive Director of the Kansas Foundation for Medical Care and has developed a sophisticated review organization; and

WHEREAS, James E. Agin has been outstanding in the excellent performance of his job; and

WHEREAS, Because of budget restraints it was necessary to release James E. Agin from his position as Executive Director in order to keep KFMC a viable organization; therefore be it

Resolved, That James E. Agin be commended for his excellence as Executive Director of KFMC.

RESOLUTION NO. 82-23

Appreciation to the KMS Auxiliary

WHEREAS, Recently a display of photographs, with small plaques showing year of service in office as President of the KMS was seen on the wall of the Conference Room of the Headquarters of KMS at 1300 Topeka Avenue, Topeka; and

WHEREAS, There are those who feel that this display may be of interest to those who meet in this room from time to time; and

WHEREAS, Certainly all members of this House of Delegates appreciate the efforts of our spouses in the Kansas Medical Society Auxiliary, and since they have no headquarters building or office of their own; therefore be it

Resolved, That we invite the members of our Auxiliary to organize and display on the wall of our Conference Room in our office building a display (similar to the one mentioned above) of photos of the past presidents of their organization.

Consultation Piece

(Continued from page 312)

fore we are confronted with that most effective of weapons — economics. Since competition and cost containment are to be ways of life, the need to utilize the most effective communication and the most economical application of these technologies will be preeminent — they are, after all, one of the prime reasons costs have increased so greatly. Much as the clinician loves the role of the knight in shining armor, the squires and grooms are going to be playing more prominent parts in the tournament. — *D.E.G.*

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OZARK CHEST CONFERENCE — The second annual Ozark Chest Conference for pulmonary physicians, general internists, cardiologists, radiologists, and thoracic surgeons will be held September 24-26, 1982, at the Osage House, Lake Ozarks, Missouri. For details contact Susan Pingleton, M.D., UMKC School of Medicine, 2411 Holmes Street, Kansas City, MO 64108.

Nail Diseases

(Continued from page 306)

Answers

1. *True*
2. *False*
3. *False*
4. *False*
5. *False*

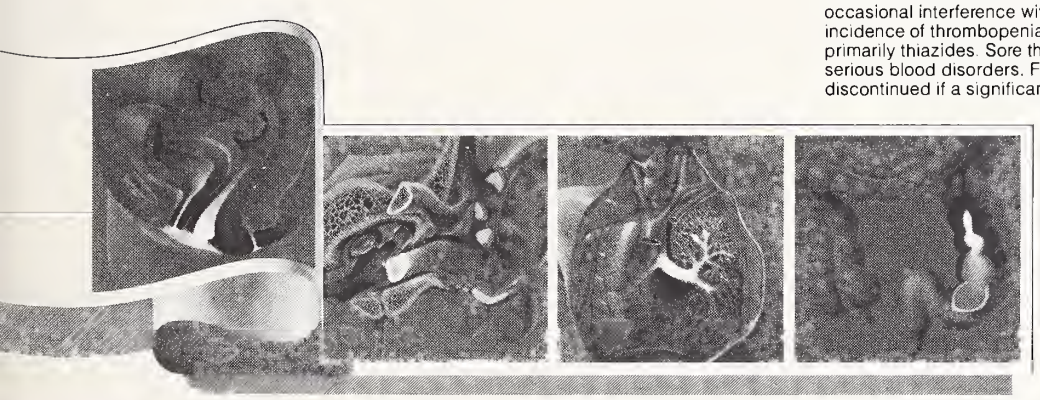
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faster relief of diarrhea than with ampicillin[2]

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. Limited clinical information presently available on effectiveness of treatment of otitis media with Bactrim when infection is due to ampicillin-resistant *Haemophilus influenzae*. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.

For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.

For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.

Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.

Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:

Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.

Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.

For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.

ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:

Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.

PNEUMOCYSTIS CARINII PNEUMONITIS:

Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

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1. Rubin RH, Swartz MN: *N Engl J Med 303*:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

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*due to susceptible strains of indicated organisms

Please see previous page for summary of product information.

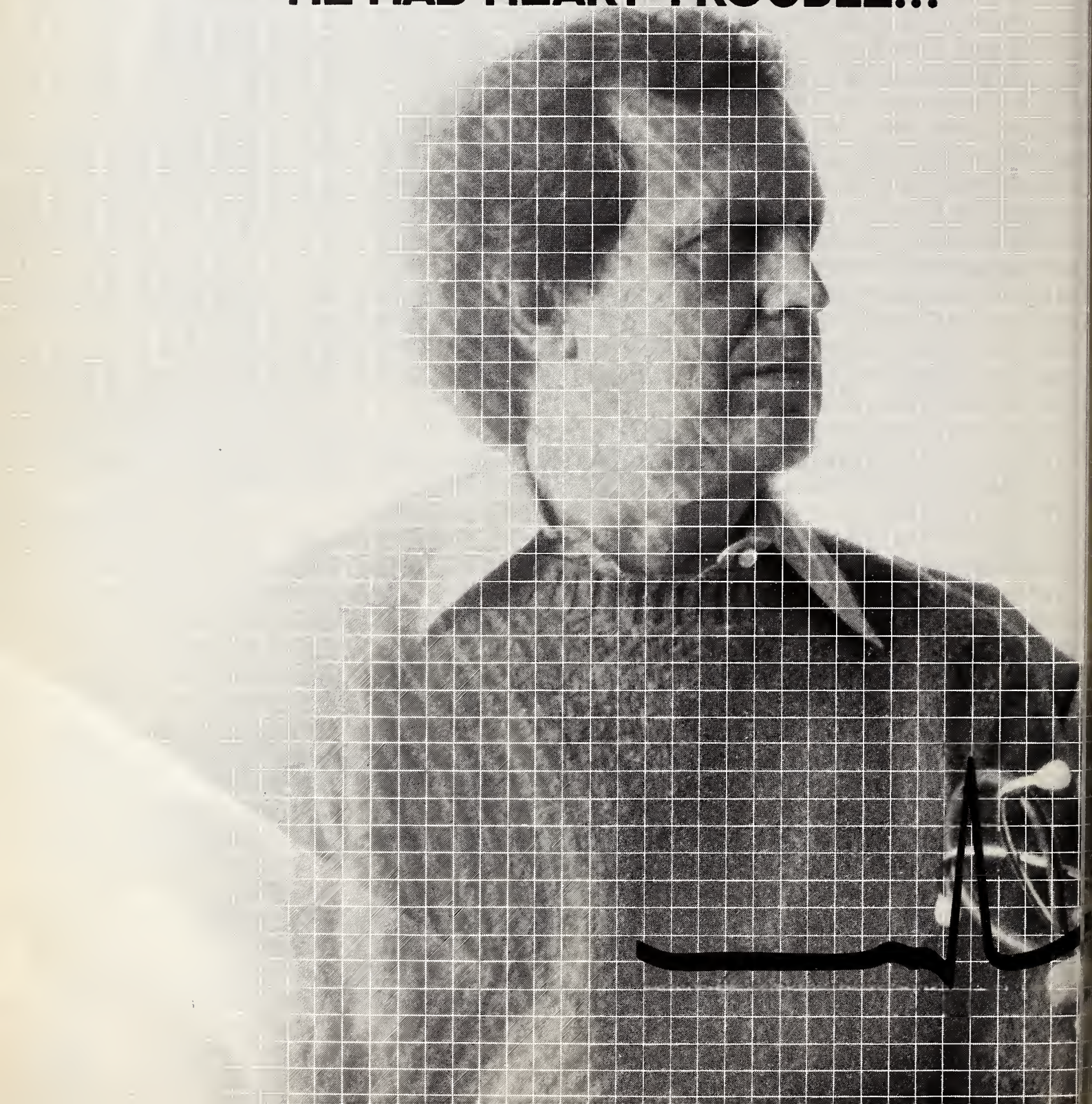
GERIATRICS

THE Journal Kansas Medical Society

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1982

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NO. VII

THE PATIENT THINKS HE HAS HEART TROUBLE...





...YOU KNOW IT'S REALLY ANXIETY SYMPTOMS

His presenting symptoms: palpitations, chest pain, chronic exhaustion and occasional difficulties in breathing. Good reason for concern. A complete workup uncovers no organic dysfunction, but it *does* reveal excessively high levels of anxiety and apprehension.

For rapid relief you prescribe Valium (diazepam/Roche)

At times like this, Valium (diazepam/Roche) can be a potent therapeutic ally. It works promptly. Within just a few hours, the patient begins to feel calmer. And in a few days, anxiety relief not only becomes more pronounced but a noticeable reduction in anxiety-generated somatic symptoms also occurs.

Equally important, Valium is generally well tolerated. Side reactions more serious than drowsiness, ataxia and fatigue are rare. Patients should, of course, be cautioned against driving or drinking alcohol while on Valium therapy. Periodic reassessment of the need for antianxiety medication should also be performed.

VALIUM® (IV)
diazepam/Roche

2-mg, 5-mg, 10-mg scored tablets

BECAUSE YOU'RE CONVINCED THE PATIENT NEEDS IT

ROCHE®

Please see summary of product information on the following page.

VALIUM®(diazepam/Roche)

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety. Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic. Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation. The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
How Supplied: For oral administration, Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100* and 500;* Prescription Paks of 50, available in trays of 10.* Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25,† and in boxes containing 10 strips of 10.†

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†Supplied by Roche Laboratories, Division of Hoffmann-La Roche Inc., Nutley, New Jersey 07110

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ROCHE PRODUCTS INC.
Manati, Puerto Rico 00701

After Quinlan

(Continued from page 375)

Restraint by all concerned will undoubtedly produce anxiety. There is human comfort in action, even if the action solves nothing, or even if it makes matters worse. Such restraint will produce inconsistencies as well, but final consistency simply is not possible at this time. All involved may have to become more comfortable knowing that health professionals will have to act at times where the morality of the situation is uncertain, where no Commandments exist. The alternative is a medical-ethical Alice-in-Wonderland, a fantasy world in which what we are saying about the sanctity of human life bears little relation to what we are doing about it.

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Register
to
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

It was a privilege to serve as Presidential Delegate to the National AMAA Convention June 13-16. Of the 345 delegates present, seven delegates and three alternates were from Kansas. Other delegates attending were: Betty Moore (Robert), Caney; Diane Sanders (Alan), Lawrence; Ann Rempel (John), Wichita; and Carol Loeffler (James), Wichita. Alternate delegates were Caryl Bichlmeier (Frank), Shawnee Mission; Kathleen Wedel (Kermit), Minneapolis; and Nancy Macy (Ted), Salina. Also attending were National Board members Lela May Young (Chester), Kansas City, past National President; and Jean Cavanaugh (Clair), Great Bend, past National AMAERF Chairman and newly elected North Central Regional Director of AMAA. We were pleased that Mrs. Young was given the honor of installing Betty Payne (Torrence), New York, as the 1982-1983 President of the AMAA.

In his speech to our Auxiliary, Daniel Cloud, M.D., President of the AMA, stressed that the major factor in accident and disease is alcohol, stating that there are 205,000 deaths each year relating to alcohol, probably half of which are preventable. He also stated that of all cancer deaths (350,000/year), 30 per cent are due to smoking. He feels more emphasis should be put on prevention of disease.

In addition to our own House of Delegates meetings, we attended workshops on membership, AMAERF, health projects, and legislation. A symposium on "Family Relationships and the Medical Marriage" was held one afternoon.

Each state president makes a report of the activities of her state organization during the past year, and we always come home with new ideas. Betty Moore gave a good report on the activities of our Kansas Auxiliary, and emphasized a baby sitting training program one county auxiliary sponsors in an effort to prevent child abuse.

We had respite from convention meetings when we attended "The King and I" with Yul Brynner. Another evening we were dinner guests of the Kansas Medical Society at the Marriott Hotel, and appreciated the opportunity to be with your leaders. Visiting the various state hospitality rooms is always enjoyable, and the Kansas room was very popular. We heard nice comments about the wheat weaving corsages given by KMS in your hospitality room.

Meldon Laury
President
Kansas Medical Society Auxiliary

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- You want to get involved with academic medicine, full-time.
- You want to subspecialize, but can't support your family on a fellow's stipend.

It's time for a change.

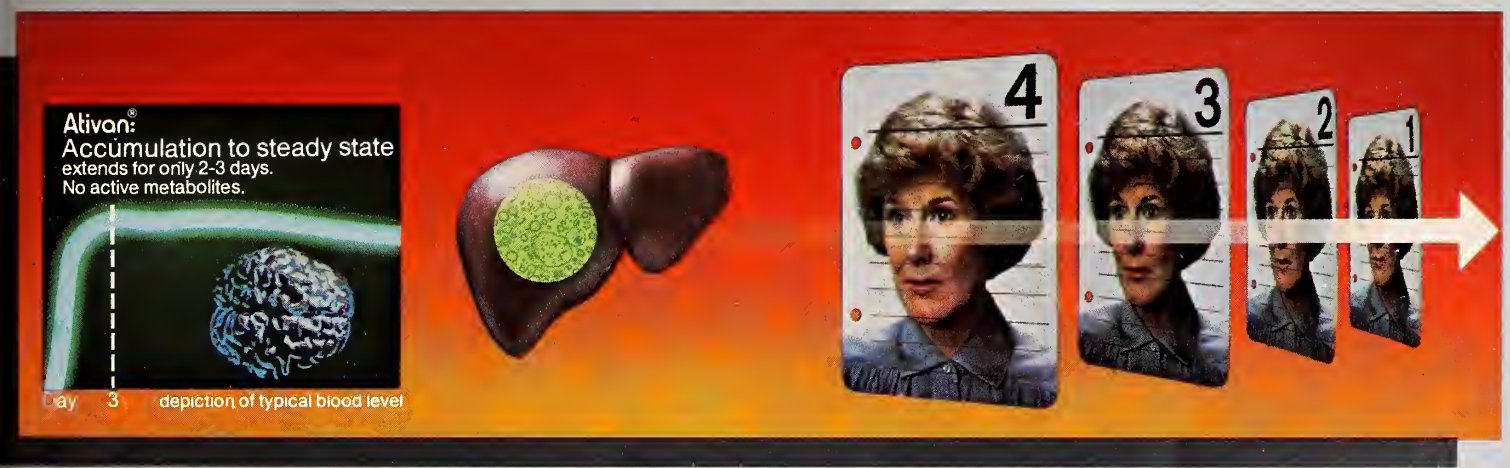
If you are seriously considering changing your situation, you owe it to yourself to consider the Army Medical Department. We have an amazingly wide variety of practice situations available to qualified physicians. Clinical and hospital-based practices in small towns, cities, major metropolitan areas. Sunbelt, Snowbelt, Europe, Asia, Panama. Full-time academic positions. Full-time research and development positions. Fellowships that pay like practice positions.

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(Inquiries held in strict confidence; position guaranteed before commitment.)

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Ativan®: (lorazepam) Agent of Change because...



shorter acting, less accumulation

Unlike most benzodiazepines, Ativan has a short half-life, no active metabolites, and accumulation to steady state extends for only 2-3 days. Ativan is therefore less likely to cause excessive sedation.*

no interaction with drugs metabolized by P450 enzymes

Because it is metabolized by simple conjugation rather than complex oxidative reactions, Ativan does not compete with other drugs for hepatic P450 microsomal enzymes. Concomitant use with Tagamet® (cimetidine), for example, does not result in delayed clearance or increased sedation†—effects which have been reported with other benzodiazepines.[1-5]

greater control of therapy

The short half-life of Ativan facilitates more rapid response to dosage adjustments, aiding you in titrating therapy to the patient's changing needs. Once you decide to discontinue Ativan, it will be out of your patient's system 4 days after the final dose—unlike the long-acting, multi-metabolite benzodiazepines which take as long as 2 weeks to be totally eliminated.

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*Pharmacokinetics cannot as yet be directly related to efficacy.
†All benzodiazepines produce additive effects when given with CNS depressants such as barbiturates or alcohol.

See important information on following page.

Ativan®
for (lorazepam) IV
Anxiety

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan®
for (lorazepam)
Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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In the treatment of insomnia

Good mornings start with restful nights.

Dalmane *(flurazepam HCl/Roche)* patients fall asleep faster, sleep longer and seldom awaken with morning hangover.

Feeling well rested in the morning usually means having slept well the night before. And for insomniac patients receiving hypnotic therapy, a good morning also means awakening with few side effects from their medication. Many physicians choose Dalmane for their patients who suffer from insomnia for this very reason.

Aside from enabling patients to fall asleep more quickly and sleep longer, Dalmane seldom causes morning hangover. Most Dalmane patients feel alert and refreshed when they awaken. In 53 paired-night clinical studies comparing Dalmane and placebo in 2010 insomniac patients with a variety of secondary diagnoses, most Dalmane patients awakened more alert and refreshed, and less groggy and drowsy, than on nights when they had taken only placebo.[1] In a double-blind crossover study of 42 patients in private practice, approximately three times as many patients reported feeling refreshed and alert upon awakening after a night on Dalmane (flurazepam/Roche) compared to placebo nights.[2] This difference was highly significant ($p<0.001$). And a retrospective study of 2542 hospitalized patients who received Dalmane revealed only a 3.1% incidence of side effects.[3]

While residual effects from Dalmane therapy are infrequent, patients should be cautioned about drinking alcohol, driving or operating hazardous machinery after ingesting the drug.

Efficacy and safety in a broad range of patient types.

Over 2000 clinical trials involving more than 10,000 patients have shown that Dalmane patients fall asleep sooner, sleep longer and experience fewer nocturnal awakenings.[4] The safety and efficacy of Dalmane have been demonstrated in medical and surgical hospitalized patients, in patients seen in office practice and in elderly patients.[5-8] Since the risk of oversedation, dizziness, confu-

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sion and/or ataxia increases with larger doses in the elderly, it is recommended that the dosage be limited to 15 mg.

Moreover, the efficacy and safety of Dalmane for the treatment of insomnia have been demonstrated in thousands of patients with a variety of primary medical conditions, including cardiovascular, neuropsychiatric, endocrine-metabolic, gastrointestinal, genitourinary, respiratory and musculoskeletal disorders.[1] Dalmane (flurazepam HCl/Roche) is contraindicated in pregnancy and in patients hypersensitive to the drug.

Avoids rebound insomnia upon discontinuation.

Rebound insomnia—a worsening of sleep beyond pretherapy levels after drug discontinuation—has been reported as a potential clinical problem with some hypnotics.[9,10] However, this problem has not been reported with Dalmane. In eight out of eight sleep laboratory studies, there were no reports of rebound insomnia.[11] When you prescribe Dalmane, you can be confident of efficacy that enhances therapeutic progress. Your insomniac patients can be assured of a restful night, night after night—a good start for a good morning.

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For efficacy from the beginning to the end of therapy

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Please see following page for summary of product information.

Dalmane®
flurazepam HCl/Roche
15-mg/30-mg capsules

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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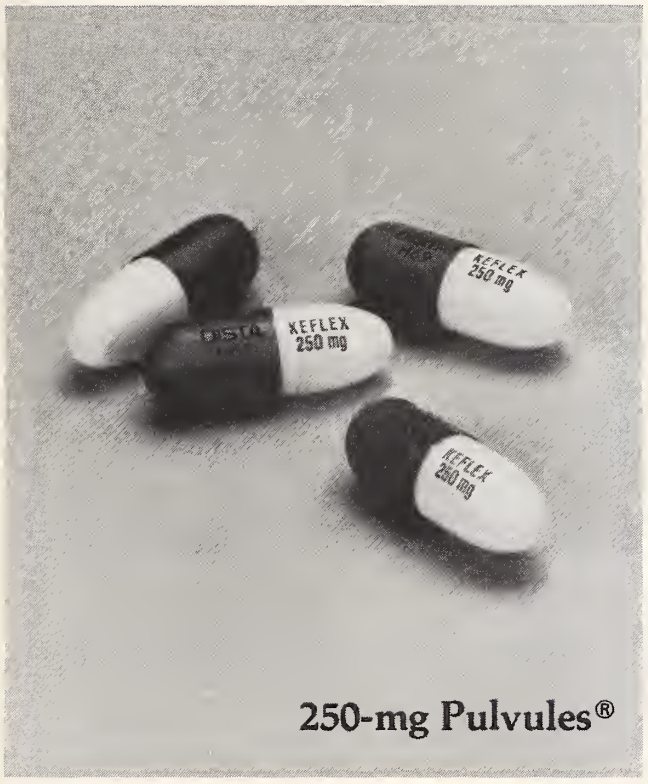
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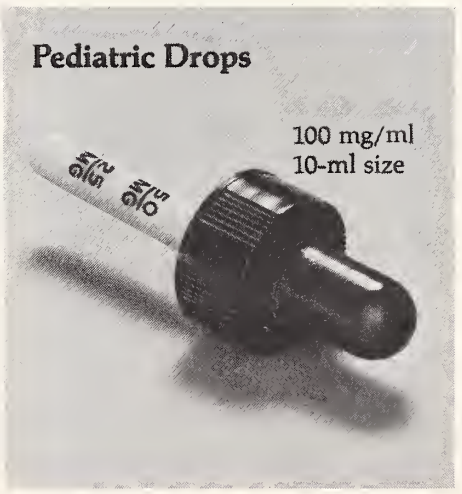
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KaMPAC MEMBERS 1982

The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

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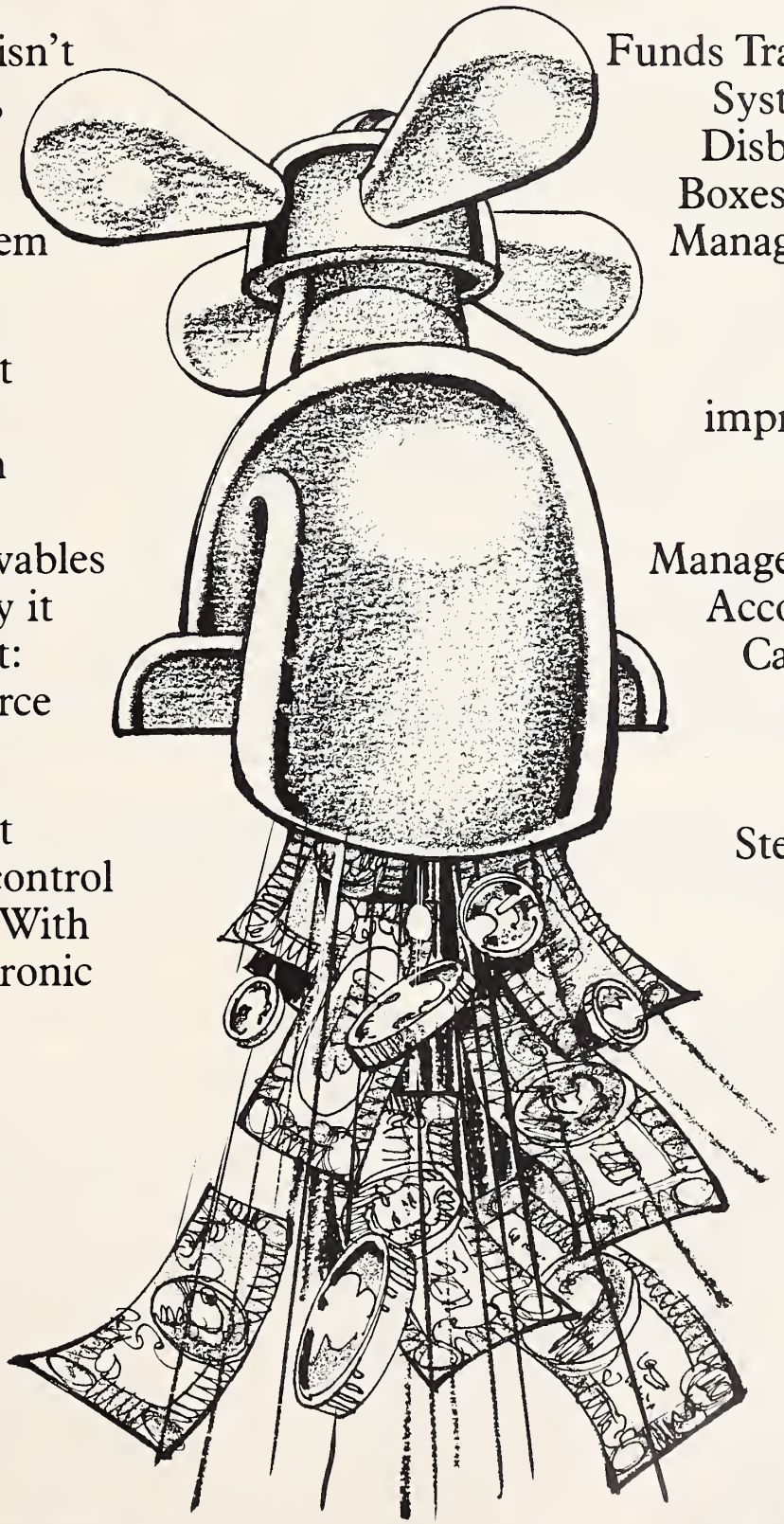
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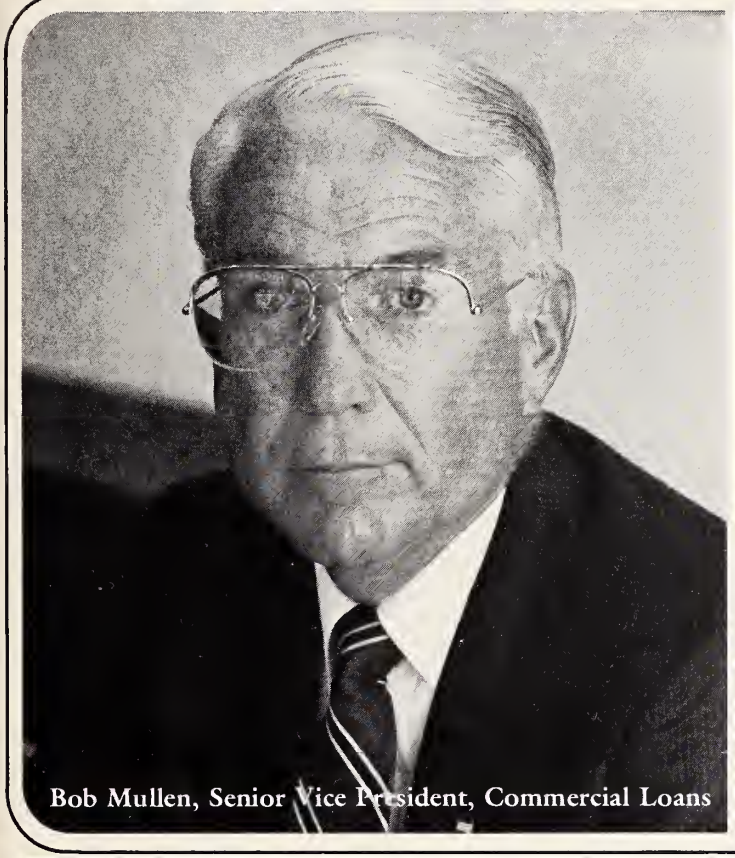
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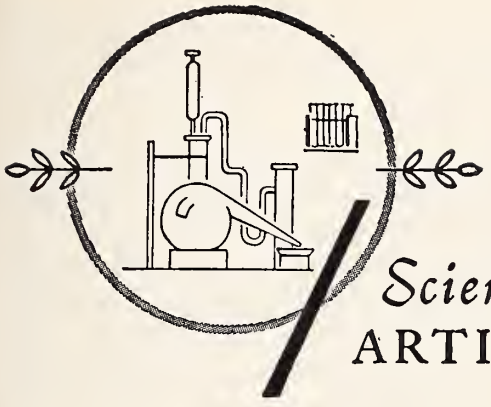
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Scientific ARTICLES

Old Age

It's Not a Disease

KENNETH P. ZABEL, M.D., ***Pittsburg***

IMPROVED HEALTH CARE for the older American is both possible and desirable. Important advancements have been made toward understanding pathological processes of the aging mind and body.

The major problem in geriatric care today is the undeniably slow process of medical schools in incorporating geriatrics and gerontology solidly into the medical school curriculum. There is an understandable lag period between the emergence of a body of information and its dissemination through the formal teaching process. However, the rapid emergence of specialties such as nephrology, immunology, and oncology raises questions as to the reasons that geriatric teaching has progressed so slowly.

Nowhere in the entire field of medicine is the need to treat the whole patient so important as in geriatrics. Important as the subspecialties are, they represent only a part of a whole. There is a need to pull all the parts together and treat the whole patient, a need to interpret the effects of a failing heart, arthritic knees, and poor memory in a given person. This information needs translation into a meaningful treatment program designed to the uniqueness of each specific human being. Even in this day of relatively sophisticated medical care there is still an urgent need for a member of the medical care team to coordinate all subspecialty areas. There is still an urgent need for someone to understand the whole regardless of how many specialists there are who can explain the parts.

This is not to advocate that geriatrics become another medical specialty, but just as a child is not a miniature adult, neither is the older person simply an overly mature individual. There are distinct physiological differences as well as unique disease processes that serve to identify the geriatric patient as well as the pediatric patient. The concept of holistic treatment of the patient is more relevant in geriatrics than in any other area of medicine.

Much can be done to improve health care for the elderly. Some diseases are incurable, but no patient is untreatable. Appropriate treatment is defined by the physical condition — not the age — of the patient. It is important for the physician to use an holistic and positive approach to the care of geriatric patients.

Who Are the Aged?

Who are the old and the aged? The simplest determinant of old age is also unfortunately the poorest, *i.e.*, the number of years since birth. Aging should not be judged in years only. To do so ignores such considerations as the maturity of the person, his/her accomplishments, contributions to society, and

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present and future potential for achievement. It deals with the quantity but not the quality of life.

Traditionally the individual over age 65 years has been classified as belonging to the older age group. The justification is more a matter of convenience than of rationale. Social security for most persons begins at age 65 years. Until recently, the mandatory retirement age for millions of Americans was age 65 years. Aging, however, is an individual process that defies simple definitions. There are enormous differences in the rate at which each of us age. Many factors determine this process; genetics, environment, and personal life style are but a few of the determinants, and each of these factors varies widely in influencing the aging process of the individual person.

The aging process varies both in time and in different societies. At the turn of the century a newborn's life expectancy was 48 years for a male and 51 years for a female. In 1974 these figures had risen to 72 and 76 years respectively. In oriental cultures, the individual gains esteem and respect with each advancing year. A province in the USSR — the Azerbajjan — has received much publicity as well as social and medical investigation because its members commonly live more than 100 years.

There are two biological determinants of aging — the mind and the body. What of the person who is confined to the wheelchair because of a twisted, lame body which cannot support his weight but whose mind remains clear and whose spirit is still nimble? Is that person really old? Or consider the unfortunate who have physiologically healthy bodies but whose minds are hopelessly and permanently crippled. Aging is not a precise concept. It is not as precisely defined as are the states of health and disease. Just as health is more than the absence of disease, aging cannot be defined as the loss of youth. The young person may not be healthy and the aged person need not be ill. Virtually every older person has one or more chronic diseases. Arthritis, heart disease, and cerebrovascular disorders are quite common. But why should a person be identified by limitations? A glass that is half empty is also half full. The older person should have an identity that is based on his accomplishments and his potential and not on the weakness of his health.

Youth is not a time of life — it is a state of mind. Nobody grows old by merely living a number of years; people grow old only by deserting their ideals. Years wrinkle the skin, but to give up enthusiasm wrinkles the soul. Worry, doubt, self-distrust, fear and despair — these are the long, long years that bow the head and turn the growing spirit back to dust. You are as young as your self-confidence, as old as your fear, as young as your hope, as old as your despair. (Samuel Ullman)

Aging and Longevity

Life expectancy cannot be defined, however careful the study of statistics. How long can the human body be expected to perform satisfactorily? Obsolescence may be built and engineered with care, but the human body was not designed to perform for a specified number of years. Most humans were created with near perfect physiologic bodies whose function is determined by the emotional and spiritual behavior and health of each person.

Most of us were endowed with an intelligence to maintain and enjoy our physical existence. It is not mandated that we become ill. Our bodies and minds are not programmed to become diseased; our bodies are not burning candles which inevitably flicker and die. We do not become less and less as we become older and older. Aging should not be defined as the loss of function but rather as accumulation of experiences and wisdom which enriches our existence.

There is a correlation between aging and the appearance of disease, to be sure. The older we become, the greater the risk for developing a disease process. It is a fact that 80 per cent of our medical services are directed toward the oldest 20 per cent of our population. But these are statistical observations that do not apply to the individual person. Many — in fact, most — elderly persons enjoy good health. Only 4 per cent of our population age 65 years or older is confined to a health care institution at any one time. Most of us can and will live out our lives in our own communities and our own homes.

Medical Treatment

The physician's expectations are an important determinant in the recovery potential of each patient. Negative attitudes of pessimism or rejection on the part of the physician are quickly recognized by the patient. On the other hand, positive expectations maximize recovery potential and are of vital importance. If the physician is willing to accept less than optimal treatment results, the patient will indeed fall short of his potential. The physician may be able to treat successfully when the patient has a basically negative outlook, but rarely will the outcome be favorable if the roles are reversed. The physician is a powerful influence, and nowhere is this more evident than in the treatment of the elderly patient.

Myths

There is wide-spread belief that incurable disease is inevitable or even pre-ordained. There is also a

feeling that when such disease occurs in the older individual, it is less responsive to treatment and that death is the inevitable outcome. The facts support none of these myths.

Some disease states, such as cancer, do occur more commonly in old age than in middle age. But some also occur more commonly in children than in middle-aged adults. In children up to 14 years of age, cancer is second only to accidents as the leading cause of death. It is therefore illogical to conclude that any disease is primarily age related. Similarly, it is illogical to conclude that disease results from some as yet undiscovered deterioration of body function.

Aging lends itself readily to interpretation as an "incurable disease," but even the fact that a disease may be incurable should not cause all hope to be abandoned. Incurable does not mean untreatable. No patient has ever been cured of diabetes or high blood pressure or — for that matter — hay fever. No one really expects to be cured of heart disease, either.

It is the physician who has been largely responsible for the prevailing attitude of pessimism or even rejection of aging in the past. S/he rejects the patient with phrases such as "I can't do any more," "There is no hope medically," or "The condition is no longer treatable." What is being said, in fact, is "Go home and don't bother me; you make me feel uncomfortable." Physicians are human and they have human weaknesses. Most are egotistical and hold a high self-image. There are reasons why this may be desirable. However, these characteristics cause them to feel frustrated — even angered — by the patient who cannot be cured. And to have a patient die is a disturbing and even threatening experience. But there is no such thing as an untreatable patient. Every patient can be given solace of mind and relief from physical pain. Every patient can be given the feeling that s/he is not facing this most difficult of all life's problems alone. Every patient can be helped in some way. The patient's family and personal physician can be strong sources of comfort. Most persons do not fear death itself as much as the anxiety over things left undone.

The physician should be responsible for bringing meaningful communication between the patient and his family. Many times a family will ask that a patient not be told the nature of the illness. If death is a more than usual certainty, whether from disease or aging, the family — and physician — are mistaken in the belief that they are somehow sparing their loved one. Nothing could be more wrong. The patient knows. He has only to look into the eyes of others and he knows. If he in turn tries to spare the family by playing this pretend game, there develops a situation where no one can lend support to anyone. The physician should not agree to become a part of this conspiracy of silence. The patient needs and deserves to know what is happening. He needs to make plans. Most of all, he needs to be able to share this burden with others. One cannot offer comfort and solace to any individual whose eyes one cannot meet. There is no fear equal to the feeling of being abandoned, alone, without anyone with whom one can express feelings. Moreover, this is a time for reflecting on the past life, for reliving the shared joys and memories. It is also a time for making future plans, planning estates, and preparing the mind and spirit for what lies ahead. Couples who have lived through and shared many of life's crises should not be apart at a time such as this. Almost every patient is strong enough to know. The real problem is families who are not equal in courage. They excuse their own inability to cope and mistakenly justify it by thinking they are sparing the victim.

Death is not a part of disease; it is a part of life. The mode of death is meaningless, the manner in which a person dies is everything. Sir William Osler was a brilliant physician of the late 1800s. He made valuable contributions in studies of the heart, infectious diseases, and infant mortality. He is best remembered, however, for his personal touch with his patients. Dr. Harvey Cushing, in his biography of Dr. Osler, relates the following:

> A mother recalls the memory of her 6-year-old daughter terminally ill with leukemia. Dr. William Osler attended her. Twice a day he visited her little room which to her was the entire world. She looked forward to his visits with pathetic eagerness. It was late summer and he would talk to her of the little animals, the trees, and the flowers. He made her room come alive with visions of frolicking bears and mythical dwarfs. All the time he was touching and probing and inspecting. In this way he would find out all he needed to know to minister to her physical and emotional needs.
>
> The most exquisite moment came one cold October morning when her strength was ebbing quietly. They had a tea party, our little girl and the great Doctor Osler. The little animals and fairies were there. The table was decorated with the last summer roses. Dr. Osler spoke of the beautiful things of this life. Like the roses, he said, little girls could not be with their loved ones forever. After their life was over, he said, the memory of their beauty would linger always. And she understood and was not sad.

Treatment

There are a number of factors that may delay and thereby compromise the relationship with the individual. Whether it is the treatment of disease or management of the aging process, these factors may

be classified as (1) patient factors; (2) family factors; and (3) physician factors.

Patient Factors

The older person is less likely to be aware of or may fail to appreciate the meaning of altered physiological functions of the body. Such changes may be the only warning of a disease state. They are often attributed to aging and their true significance is not recognized so that diagnosis and treatment are delayed. Sometimes older patients are reluctant to report to family or physician symptoms that should be investigated immediately. They may fear that they are too self-conscious or that their complaint is really too minor to be significant. Weight loss or low grade fever may be signs of various diseases. Pain may be absent in serious illness or present with no definable disease.

The most unfortunate circumstance is the patient who fears he may not be taken seriously or may even be subject to ridicule, and one of the greatest causes for delay is fear itself. The patient either knows or strongly suspects the real significance of his symptoms. Even obvious signs are hidden from others. The procrastination may be rationalized that nothing can really be done anyway, or may take the form of true denial. The patient may fear separation from home or family. He may envision financial ruin or a long and painful treatment course that is likely to only prolong what he feels will be the ultimate event.

Most pathetic is the patient who fails to seek or even denies treatment because of age. This is the "Well, I've lived long enough" or "I'm ready to go when God wills" attitude. A person is always too young to die of a treatable disease. A needless death is a tragedy at any age.

Family Factors

Virtually everything in our society becomes more valuable with age — everything, that is, but people. Antique furniture, cars, toys, and even comic books — the older the better. We take pride in restoring and preserving. But what of people? Are old people also not invaluable? Who sells experience? Where can loyalty and years of service be bought?

The family may be responsible for delaying medical attention. This is particularly likely to happen when decision making is necessarily assumed by a younger member, usually a son or daughter. The desire to spare the parent from the discomfort and anxiety associated with diagnostic studies or treatment methods is the most common reason for delay. In some cases the assumption is made that the age of the patient contradicts the treatment that is indicated. Such judgments are usually based on emotional considerations rather than facts. There are very few instances where such delay will prove to be in the best interest of the patient. Usually, delay results only in more extensive procedures at a later stage of the disease with less favorable outcome.

Physician Factors

The physician may be responsible for delayed diagnosis and less than optimal treatment of the older patient. The reasons are varied, but in general they reflect the prevailing attitudes and values of our society. This results in less prestige in treating older patients and fewer physicians with an interest in this area.

Age alone should not be a factor in considering treatment methods or alternatives. Even patients of advanced age can be operated on with acceptable risk. In a series of 272 patients over the age of 90 years operated on at Los Angeles County Hospital, less than 5 per cent died in the immediate postoperative period. This low rate was in the group of patients without serious associated medical disease. However, in those patients with serious associated pulmonary disease, 63 per cent died following surgery. It is clear, then, that the presence of disease, but not old age itself, is the determining factor in decision making regarding treatment. This point needs to become more widely appreciated, by the medical profession in particular.

Conservative treatment is ordinarily a virtue. If medical treatment can achieve a comparable result and avoid the risks attendant with surgery, then the choice is easily made. Conservatism should not be confused with indecision or with compromising treatment results because the distinction between an aged organ and a diseased organ is not appreciated. Conservative treatment may be radically wrong. Delay of proper treatment and unjustified compromise resulting in less than optimal outcome will inevitably ensue if the physician acts in haste or with inadequate data. The physician should teach by example. A thorough, considerate, and compassionate approach succeeds in the older patient just as it does in the younger patient.

Perspective in Hypertension

Diagnosis and Treatment of the Elderly Patient

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HYPERTENSION in the elderly (defined as age 65 or greater) is a common clinical problem and may be systolic-diastolic hypertension or "pure" systolic hypertension.

Since blood pressure is a continuous variable throughout life, the definition of hypertension is somewhat arbitrary. In most studies, however, elderly patients are considered hypertensive if the diastolic pressure equals or exceeds 95 mm Hg and the systolic pressure equals or exceeds 160 mm Hg. Diastolic hypertension may be further categorized as "mild" or "borderline" (90-104 mm Hg), moderate (105-114 mm Hg), and severe (115 mm Hg or greater).[1]

Epidemiology and Prevalence

In Western industrialized nations, including the United States, a continuous rise in average systolic blood pressure occurs with age. Average diastolic pressure, on the other hand, increases until approximately age 55 years, then begins to decline. In some primitive societies where weight does not increase with age and there is minimal salt intake, individuals remain normotensive throughout life.[2]

The prevalence of hypertension, as defined, in those 65-74 years of age is in the range of 35-50 per cent. In general, blacks have a greater prevalence than whites.[3] It should be noted that some of the data on which these figures are based are from a single blood pressure determination. If only half of these subjects were hypertensive on subsequent examination, the numbers involved would remain enormous.

Morbidity and Mortality

The Framingham study has demonstrated that hypertension is a major risk factor for cardiovascular disease, including coronary artery disease, heart failure, and stroke at all ages. Hypertensive subjects between 65 and 74 years had an annual incidence of cardiovascular disease three times greater than normotensive subjects in the same age group.[4]

The relationship of "pure" systolic hypertension to morbidity and mortality remains in question, although there is evidence of an associated deleterious effect. Gifford believes the correlation is obvious from available data, although he notes this may not be a cause and effect relationship.[5]

Hypertension in the elderly, a common problem with a variety of unique features, may represent pre-existing hypertension or may arise *de novo*. There are numerous problems related to diagnosis and treatment of hypertension in this age group that are reviewed herein.

There is general agreement that hypertension places the elderly patient at greater risk for cardiovascular morbidity and mortality, and that this risk increases with increasing age and increasing blood pressure.[6]

Pathophysiology

Most systolic/diastolic hypertension in the elderly is, as in younger patients, idiopathic or "essential," with a hemodynamic pattern characterized by high peripheral vascular resistance and low plasma volume. These two factors are important therapeutically since they may increase sensitivity to neurogenic blockade. Plasma renin tends to be low in spite of decreased plasma volume. There does not appear to be any clear association of the renin-aldosterone mechanism with essential hypertension in the elderly.[3, 6] Baroreceptor sensitivity is decreased with a resultant impairment of buffering capability over wide ranges of blood pressure.[6]

Isolated systolic hypertension is usually associated with decreased aortic distensibility due to atherosclerosis which leads to the disproportionately high systolic pressure. Other causes, *e.g.*, aortic insufficiency, are much less common.

Evaluation

Most systolic/diastolic hypertension in the elderly is "essential," with less than 5 per cent of cases secondary to a defined pathologic process.

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History. The history should emphasize detection of possible end-organ damage. Symptoms of angina, congestive heart failure, renal disease, and cerebrovascular disease should be sought. Causes of secondary hypertension should also be explored, *e.g.*, palpitations, sweating, headache (pheochromocytoma), weight gain, hirsutism, glucose intolerance (Cushing's syndrome). A detailed drug history, including such medications as steroids, estrogens and nosedrops, is essential. With pure systolic hypertension, causes other than aortic atherosclerosis, such as the high output states of hyperthyroidism, Paget's disease, anemia, and aortic insufficiency should be sought.[3]

Physical Examination. Physical examination should include lying, sitting, and standing blood pressures in both arms. It is particularly important to note orthostatic blood pressure changes after the patient has been standing for 30-60 seconds. The character of the pulse may be full and bounding in systolic/diastolic hypertension. A collapsing pulse may be seen in isolated systolic hypertension. The fundi should be examined for hypertensive changes. There may be signs associated with the high output states previously mentioned, *e.g.*, goiter or diastolic aortic murmur. Abdominal bruits and aneurysm may be detected.[3]

Laboratory. Laboratory examination need not be extensive. Basic examination includes a CBC, urinalysis, electrolytes, creatinine and BUN, cholesterol and triglyceride, serum calcium and phosphorus, and an EKG. Some would include a chest x-ray. This will suffice for the majority of patients. Libow[3] has suggested further workup in the following circumstances:

- Diastolic pressure above 115 or 120 mm Hg.
- Advanced retinopathy, such as grade III or IV.
- Recent onset of diastolic hypertension or marked further elevation of blood pressure in a known elderly hypertensive patient.
- Persistent low serum potassium level without kaliuretic medication (diuretics, L-dopa) or licorice ingestion (suggesting hyperaldosteronism).
- Signs or symptoms suggestive of pheochromocytoma.
- A diastolic pressure in the range of 105-115 mm Hg in a patient who does not respond to standard treatment.

Further suggested steps are:

- Rapid sequence intravenous pyelogram (renovascular hypertension).
- Urinary aldosterone (hyperaldosteronism).
- Urinary vanillylmandelic acid (pheochromocytoma).
- Renal angiography — only if surgery is contemplated.

Rationale for Treatment

The association of hypertension with morbidity and mortality does not necessarily imply that treatment will be beneficial.

The Veterans Administration Cooperative Study clearly demonstrated a decrease in morbidity related to stroke, heart failure, and renal failure in treated male patients 60-74 years of age with diastolic blood pressure above 105 mm Hg. A similar decrease in coronary artery disease was not observed. For diastolic pressures between 90 and 104 mm Hg, benefit from treatment was suggested, but the data were not statistically significant. Females and those over 75 years were excluded from this study.[7]

The Hypertension Detection and Follow-up Program (HDFP) studied mortality in a large number of hypertensive patients and found, for the first time, a benefit in terms of decreased mortality for treated black men and women and white men aged 65-74 years with diastolic pressures of 90-104 mm Hg. No such benefit was observed in white women of the same age and blood pressure levels.[7]

Libow, however, has urged caution in the treatment of mild hypertension in the elderly and points out studies from NIH and Duke suggesting mild hypertension may have a protective effect on mentation in the elderly.[7]

The data indicate an association between isolated systolic hypertension and cardiovascular morbidity and mortality, without evidence currently to suggest that treatment is beneficial in this group of patients. The question exists whether this morbidity and mortality is due to the arteriosclerotic process which leads to the rigid aorta and thus systolic hypertension, or to the elevated systolic blood pressure itself.[5]

Treatment

Treatment of hypertension in the elderly may be guided by the admonition to "start low and go slow."

In the initial treatment of mild hypertension, many would recommend such non-drug therapy as weight loss, salt restriction and exercise, although the benefit of these measures is uncertain in the elderly and present compliance difficulties since changes in lifestyle are required.

Consideration should be given to altered drug effects in the elderly. Changes in liver and kidney function may lead to increased drug levels in some instances and greater orthostatic hypotensive effects may be observed. As noted earlier, the elderly

hypertensive tends to have a small plasma volume and a high peripheral resistance which may make them particularly sensitive to adrenergic blockers and diuretics.

A stepped-care approach to treatment may be used in the elderly as in any other hypertensive.

Step One: A thiazide diuretic in low dose is generally used. Hydrochlorothiazide in a dose of 25 mg/day is used initially and may be doubled if necessary in four to six weeks. This treatment will suffice in many patients, is inexpensive, and its simplicity enhances compliance. The patient must be watched for hypovolemia and hypokalemia. The prevention of hypokalemia is of particular importance in the patient on digitalis. Hypokalemia may be treated, if necessary, with potassium supplements, or the addition of a potassium sparing diuretic such as triamterene or spironolactone. Impaired baroreceptor response may lead to orthostatic hypotension. Hyperuricemia may occur, but is usually not clinically significant. It may be treated with allopurinol if the uric acid level is above 12 mg/dl.[6] Metolazone or a loop diuretic such as furosemide may be used if renal function is significantly impaired. Some would start with a beta-blocker such as propranolol as Step One and add a thiazide if needed, although elderly patients may not respond well to beta-blockers as a first step.

Step Two: Methyldopa, clonidine, and reserpine may each be used as second drugs. The objection to these is that they cause depression of the central nervous system, although this is often not a significant problem and these drugs have been widely and successfully used. The most problematic is reserpine because of the significant incidence of depression. Methyldopa may be started at a dosage of 250 mg/day and increased every four to eight weeks up to 500 mg twice daily. Drowsiness, dryness of mouth, depression, and orthostatic hypotension may occur. Clonidine is similar in action and potency to methyldopa, but is less likely to cause orthostatic hypotension. It, too, may result in drowsiness and dry mouth. The starting dose is 0.1 mg and may be increased every four to six weeks to a dose of 0.6 mg/day.

A beta-blocker, such as propranolol or metoprolol, may be used as a Step Two drug. Congestive heart failure and aggravation of asthma or chronic obstructive pulmonary disease (COPD) must be guarded against by careful history taking and cautious administration of these drugs. A more cardioselective drug such as metoprolol may be better tolerated in patients with COPD and asthma. Maximal doses of these drugs are not well defined, with doses ranging from 160 mg/day to 450 mg/day or higher, based on clinical response.

Step Three: Hydralazine and prazosin are the two major vasodilator drugs in use today. Hydralazine acts on vascular smooth muscle to cause dilatation with a concommitant increase in cardiac output and pulse rate.[6] It is often well tolerated in the elderly since the reflex tachycardia seen in younger patients may be blunted due to decreased baroreceptor activity. Angina may, however, be aggravated in some patients. Orthostatic hypotension usually does not occur. The lupus erythematous-like syndrome that may be associated with hydralazine usually does not occur at doses below 200 mg/day.[6] Hydralazine dosages may be increased from 25 mg/day up to 100 mg twice daily.[5] The vasodilator prazosin may also be used as a Step Three drug with caution for frequent syncope at the beginning of therapy.[7]

Step Four: Guanethedine should probably be avoided in the elderly due to its many side effects and interactions.[3]

The goal of treatment should be moderate, with acceptable pressures in the range of 150/90 to 160/95 mm Hg.[4] This should be achieved with minimum expense, side effects, and risk. Surgery for atheromatous hypertension should be undertaken only if medical treatment is unsuccessful and with an understanding of limited (approximately 35%) favorable response following correction.[3]

Comment

Current data favor treatment of hypertension in the elderly with two exceptions: (1) white females with mild hypertension, and (2) isolated systolic hypertension. Further research will be required to demonstrate appropriateness of therapy in these instances. The treatment of any elderly patient's hypertension should be approached with a conservative plan for diagnosis and therapy.

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Geriatric Hypertension

The Challenge of Personal Physician Commitment to Effective Management

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THE ELDERLY have been considered a separate age group with regard to hypertension only in the last ten years. There is no commonly understood definition as to who the elderly are. Herein, the terms "aged" and "elderly" will be used interchangeably to mean someone over 65 years of age.

Chronological and physiological ages are not synonymous. It is not surprising to note upon necropsy that many persons age 50 years have the brain tissue and blood vessels of an individual of 75 years or older and vice versa. If one falls prey to the error of viewing all 70-year-olds as the same age, the clinician will err in evaluating and treating high blood pressure.

There is no common understanding as to what constitutes hypertension in our geriatric population. Some health care providers question the benefits achieved by treating the elderly.[1] Treatment is more difficult from both psychosocial and physical perspectives and also from physiological constraints which may be imposed by impaired renal, liver, and cardiovascular function.[2] Hence, the clinician and the older patient are frequently faced with the choice of accepting the risks of hypertension or coping with the more immediate, and often disabling, side effects of adequate antihypertensive therapy.

Definition

The level at which a physician would diagnose hypertension is not the same in the 20-year-old and the 75-year-old. Since blood pressure generally increases with age, it is often difficult to clearly establish when an older person's blood pressure is pathologically high. In any age group, increased blood pressure is purely a statistical concept.[1] Thus, patients whose blood pressures are greater than two or three times the standard deviation above the mean for their age group are considered hypertensive.

Hypertension in the elderly can be defined arbitrarily as a systolic pressure above 160 mm Hg and/or diastolic pressure above 95 mm Hg. It is assumed that a borderline hypertensive will have a blood pressure of approximately 140/90 mm Hg. Many clinicians, however, define elevated blood pressure as 160-180 mm Hg systolic or "a person's age plus 100."

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Effective treatment of the elderly hypertensive presents a unique challenge that requires a personal physician commitment to recognition of the unique aspects of evaluation and treatment of senior citizens. Attention must be focused on the patient's personal historical profile, adherence to regimen, and the efficient utilization of the health care team.

To complicate matters, as arteries harden with age there appears to be an increasing gap between blood pressure values measured by the cuff applied externally and simultaneous measurements by the interarterial route.

Goals

The elderly individual must be considered as a person and not as a series of end organs affected or to be affected by the disease. The physician must examine appropriate therapy within the context of quantity and quality of life.

Both are serious considerations for a 65-year-old. Quality of life must be a main consideration in the very old, with stated goals designed to prevent recurrent ravages of cerebral vascular accidents and coronary artery crippling diseases. Goals for any well-managed hypertensive regimen would include reductions in morbidity and mortality, and an improvement in the quality of life. Prevalence and risk factors involved in the treatment of hypertension must be reviewed.

Approximately 40 per cent of white and more than 50 per cent of the black population over 65 years have either isolated systolic or systolic/diastolic hypertension.[1] A reading of 160/95 mm Hg statistically doubles the risk of death in men and increases the risk in women 2.5 times. Borderline hyperten-

sion (140/90-160/95 mm Hg) increases the risk of morbidity and mortality in men 1.5 times and doubles the risk in women. Isolated systolic hypertension greater than 180 mm Hg increases the risk of stroke and of death from coronary disease 2.5 times in persons aged 65-74 years.

Hypertension may also be a causative factor in cases of senile dementia due primarily to vascular disease. However, there is no evidence that early appropriate treatment of hypertension can prevent senile dementia.

Data from the National Institutes of Health[3] longitudinal studies on human aging and from longitudinal studies of the elderly at Duke University suggest that minimally increased levels of systolic and diastolic blood pressure within the so-called "normal" or "borderline" range may be of protective value in the elderly.[4, 5] These studies demonstrate a direct correlation between increased EEG activity and increased blood pressure. At Duke University, older individuals whose diastolic blood pressures ranged from 96 to 105 mm Hg were given repeated IQ tests during a ten-year period. The study subjects' scores improved in certain areas, while the IQ scores of a matched group of normotensive individuals remained unchanged.[6]

Preventing increased damage to blood vessels is critical, yet there is a question as to whether or not treatment of hypertension will prevent arterial thrombosis, injury, or spasm. It is the responsibility of physicians to suggest to the patient that he or she receive the benefit of the doubt.

One must not be too hasty and accept statistical evidence that casts doubt on the value of effective hypertensive management. Often biostatistics are the result of cold tabulation of numbers, divorced from the behavioral aspects of the elderly. The alternative to providing management is a struggle to improve the health of an individual whom hypertension has rendered a neurological and cardiovascular cripple.

Responsibility for Effective Management

A physician must assume the major responsibility for evaluation and coordination of all efforts in a patient's total care. However, other members of the health care team can gather data to reduce demands on physician time. Sympathetic staff can provide education, uncover adherence problems, suggest solutions, and monitor progress toward established goals. If desired, the non-drug portion of the total therapy package can be organized and implemented by paraprofessionals.[7, 8]

Treatment Process

A useful process outline would include the following features:

- A careful history, including past and present diseases and treatment; notation of sight, hearing, and physical handicaps.
- A patient profile including address, telephone at home and work (if applicable); career history; nutritional data; habits such as smoking, alcohol consumption, sleep patterns; exercise patterns;[9] hobbies and avocations; support systems; financial status; home environment; available community services.
- Physical, laboratory, and mental status evaluations.
- Definitions of specific problems and diagnoses.
- A plan of therapy including patient education, nutritional advice; pharmacotherapeutics; exercise for physical rehabilitation; stimulating, pleasurable activities for psychological rehabilitation.

A careful monitoring of the effects of a therapeutic plan is essential, as is an evaluation of the results.

A history should focus on the differential diagnosis of pure systolic hypertension or systolic/diastolic high blood pressure. This includes those conditions (in addition to age) that increase blood pressure, such as the high cardiac output in hyperthyroidism, arteriovenous fistula, Paget's disease of the bone, hyperkinetic heart syndrome, anemia, aortic insufficiency, and fevers of various sources. More often than not hypertension is a symptomless problem, but questions focusing on symptoms such as headache, dizziness, angina, intermittent claudication, and renal problems are appropriate. Medications such as salt-retaining steroids (including estrogens and corticosteroids) as well as excessive licorice ingestion may induce hypertension.

Occasionally, an older person will discuss a history of hypertension, but repeated measurements will not indicate hypertensive levels. This is important to note, in that it may reflect lability of blood pressure, the effects of previous treatment, or possibly even a recent myocardial infarction.

The patient profile is the most important aspect of the documented history for an older person.[10] It is often the basis upon which a successful plan can be implemented and good adherence assured. Personal non-medical information can provide the physician with clues as to how the patient can take part in health care maintenance.

Evaluating the patient's support system can be vital. Who would s/he contact in case of medical

emergency? Is s/he living alone? What is the nature of his/her immediate and extended family? Do they hinder or enhance the patient's living pattern?[8] The clinician should also be sensitive to the financial status of the individual. A fixed income may limit health insurance benefits and discourage the patient from filling prescriptions or keeping appointments.

Physical Evaluation

Blood pressure measurements should be taken in both arms with the patient alternately supine, sitting, and standing. The patient should be standing 30-60 seconds before standing blood pressure is taken to provide an opportunity for orthostatic abnormalities to occur. If the patient has an initial elevated reading, three blood pressure checks should be made per visit.

Physical evaluations should include careful descriptions of the nature of the pulse, fundi, abdomen, and cardiovascular system. The use of brief mental questionnaires is indicated,[1] because the results have implications for treatment and patient compliance. A careful neurological evaluation is mandatory, as speech, visual, sensory, and motor changes may reflect an existing cerebral vascular deficit.

Laboratory Investigation

Routine laboratory investigation should include the following tests: hemoglobin, hematocrit, white blood cell count; urinalysis; baseline serum electrolyte levels for sodium, potassium, chloride; serum glucose, blood, urine, nitrogen and creatinine values; cholesterol, calcium, phosphorus and alkaline phosphatase; triglycrides, high density lipoproteins; electrocardiogram; and chest x-ray if indicated.

Generally, extensive diagnostic workups are not recommended unless abrupt exacerbation of stable hypertension or sudden onset of hypertension after the age of 65 years suggests a secondary cause.[1, 11]

Extensive laboratory investigation is unwarranted except under the following conditions, most of which are quite rare:

- Diastolic pressure above 115-120 mm Hg.
- Advanced arteriovascular changes such as grade 3 to grade 4.
- Recent onset of significant diastolic hypertension or marked further changes in known hypertensives.
- Persistent low serum potassium levels with kaliuretic medication (diuretics, L-dopa) or licorice ingestion.
- Signs or symptoms suggestive of pheochromocytoma.
- Diastolic pressure of 105-115 mm Hg in a patient who does not respond to standard treatment.

Steps in the more extensive workup for the above listed indications include rapid sequence intravenous pyelogram (IVP), urine vanillymandelic acid (VMA), "catechol pathway," and renal arteriography.

Since 95 per cent of hypertension in the elderly is "essential" (*i.e.*, of unknown cause), investigation and therapy should be directed mainly toward assessing the effects rather than the causes of hypertension.[1, 11]

In severe malignant hypertension, renal function can be preserved by antihypertensive treatment. The physician must weigh the risks of morbidity and mortality associated with surgery to relieve renal vascular lesion with the benefits. The decision must be made in relation to the individual's general emotional and physical status, and anticipated outcomes.

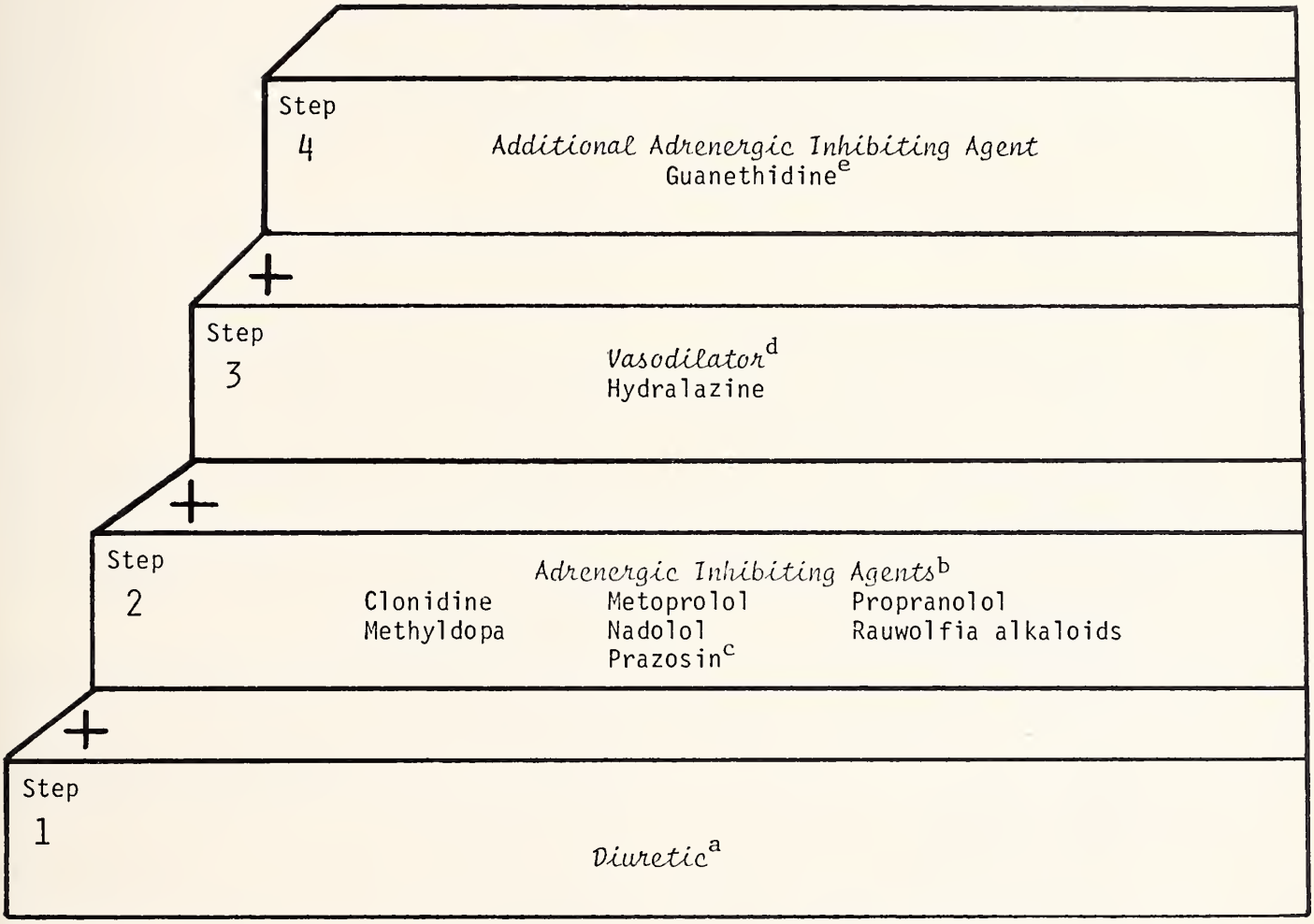
Therapy

A plan of therapy for the older person varies considerably from the regimen for a younger person with similar hypertensive problems. When deciding whether, when, and how an elderly person should be treated, several questions must be addressed. How meaningful are blood pressure evaluations at the moment or over an extended period of time? At what level of elevation should treatment be instituted and which drug should be used? What specific or general side effects might the elderly experience that are not generally present in younger generations? What effect, if any, does treatment have on longevity? How does appropriate therapy affect quality of life?

Treatment of hypertensive vascular disease can be reduced in its simplest form to two major components: "non-drug" and "drug-related" therapy.

Non-Drug Therapy

Behavior is currently being acknowledged as an important factor in the control of hypertensive vascular disease. Epidemiological studies show a strong association between blood pressure and body weight, as well as between blood pressure and sodium intake. A few studies on the effects of dietary intervention suggest that weight reduction or moderate control of sodium intake may lower blood pressure in some individuals. Both measures should be considered as an adjunct therapy for all hypertensive patients. A weight reduction to 10 per cent above or below normal weight and a sodium intake



[a]Thiazide-type diuretics are drugs of choice. Loop diuretics are reserved for selected patients.
Potassium-sparing agents may be used in combination with thiazide diuretics.

[b]Adrenergic inhibiting agents are listed in alphabetical order. This does not indicate preferential order of usage.
Clinical experience with Nadolol is limited.

[c]The postsynaptic alpha-receptor-blocking effects of prazosin appear to be more prominent than its vasodilator effects, thus encouraging its inclusion as a Step 2 drug. Prazosin may be used as a Step 3 drug if it has not been added in Step 2.

[d]Minoxidil is reserved only for selected patients and should not be considered a Step 3 drug.

[e]Guanethidine is a potent agent but may be used in small doses as a Step 2 agent.

The 1980 Report of the Joint National Committee
on Detection, Evaluation, and Treatment of High Blood Pressure

Figure 1. Step regimen in use of antihypertensive drugs: *Step 1*. Diuretics with medium duration of action such as thiazides are initiated for both systolic, diastolic, and isolated systolic high blood pressure. *Step 2*. Introduction of such drugs as propranolol, reserpine, methyldopa, or clonidine, plus a diuretic. Metoprolol tartrate, nadolol, and prazosin hydrochloride may be added or substituted. *Step 3*. Clonidine, methyldopa, reserpine or propranolol, plus a vasodilator such as hydralazine. *Step 4*. Guanethidine (seldom indicated or needed). Please refer to *Table I*, Managing of Hypertension.

of 5 gm/day (equivalent 85mEq sodium intake) has been very effective. Extended nutritional counseling is necessary for the elderly patient. Because of the loss of taste sensation, the replacement of sodium with potassium salt substitutes must be taught and monitored. Behavioral methods of hypertensive management — such as biofeedback, psychotherapy, and relaxation — are still experimental and are very difficult to implement in the geriatric population.[7]

Drug Related Therapy

All patients with moderate and severe hypertension should be treated with antihypertensive drugs. Most elderly can be treated with a minimum of side effects if the proper drug is chosen and if the dosage is increased very gradually over a period of weeks rather than days. The low blood volume, increased blood element concentration, and the reduction in baroreceptor reflex activity frequently found in people over 65 years of age may serve to increase responsiveness to hypertensive agents.

Elderly patients may be more susceptible to hypokalemia from oral diuretics if their diet is inadequate. This risk can be minimized by dietary changes and potassium supplementation, possibly through use of a high-potassium salt substitute.

Generally speaking, although complicating conditions and the accompanying need for multiple medications may present special management problems, they should not preclude treatment of the hypertensive elderly. Patients should be encouraged to bring all prescription and over the counter (OTC) medications at each visit to facilitate identification. These patients may require additional counseling to deal with adherence problems.

Complications such as congestive heart failure, systemic artherosclerotic heart disease, or cerebral vascular disease make reducing blood pressure more urgent. The presence of diabetes mellitus does not rule out the use of antihypertensive drugs (including oral diuretics), but it does require appropriate dietary changes and dosage adjustment of hypokalemic agents. Similarly, gout is not a contraindication if the gout is properly managed. Reduced renal function may frequently be a consideration in selecting a therapeutic agent.

Certain side effects such as hypokalemia or hyponatremia occur more frequently in the elderly. Drowsiness or mental confusion can usually be managed by appropriate changes in diet and drug regimen, and rarely require stopping therapy altogether. Careful attention to the reduction of the initial doses and a more gradual increase in medication amounts may help eliminate side effects. Drugs that produce orthostatic hypotension (such as guanethidine) should be avoided unless blood pressure remains at high levels. The National High Blood Pressure Education Program Coordinating Committee[8] has approved recommendations that would serve as suitable guidelines for hypertension management in the elderly.

Generally speaking, the ''stepped care'' approach is appropriate in the management of the aged hypertensive. This involves initiating therapy with small doses of antihypertensive drugs, increasing the dosage, then adding one drug after another in a sequential manner, as needed. This approach allows the clinician to take advantage of the synergistic interaction of various agents, which may permit smaller doses of individual drugs. A suggested step regimen is shown in *Figure 1*. Side effects commonly experienced have been recognized and tabulated for easy reference with special consideration as to causative factors *(Table I)*.

Before proceeding from one treatment step to another, the physician must address the possible reasons for lack of responsiveness. These include poor adherence to therapy, insufficient doses of current drugs, excessive sodium intake or weight gain, and the concomitant use of competing drugs such as vasopressor decongestants or appetite suppressants. Occasionally, a ''step-down'' process may be cautiously tried if blood pressure appears to be easily controlled. At all times, side effects, patient inconvenience, frequency of drug administration, and cost effectiveness must be considered in the therapy plan. It is important that at times of surgical intervention or trauma the treating physician be apprised of the medications the patient is taking.

Therapeutic Requirements

A carefully outlined plan of patient education plays a significant part in the success of a therapy plan. It is critical that education be based on the level of patient understanding. It is here that we value the aid and input of supportive members of the health care team. Many of our elderly develop a comradery relationship with members of the health care team, which facilitates adherence with a directed treatment program.[8]

Guidelines For Care

- Discuss with the patient what to do and how to do it. Be specific. The initial session should be limited only to the task(s) essential to achieve high blood pressure control.

TABLE I
SIDE EFFECTS OF AND PRECAUTIONS WITH COMMONLY USED ANTIHYPERTENSIVE DRUGS

Drugs	*Common Side Effects*	*Special Considerations*
Diuretics		
A. Thiazide and thiazide derivative diuretics	Hypokalemia, hyperuricemia	Digitalis and hypokalemia, gout, renal insufficiency, pancreatitis
B. Loop diuretics	Hypokalemia, hyperuricemia	Excessive diuresis, hyponatremia, ototoxicity (IV), gout, digitalis and hypokalemia
C. Potassium-sparing agents		
Spironolactone	Hyperkalemia, lethargy, gynecomastia, mastodynia, G.I. symptoms, menstrual irregularities	Hyperkalemia, renal failure
Triamterene	Hyperkalemia, nausea, weakness, leg cramps	Hyperkalemia, renal failure
*Adrenergic Inhibiting Agents**		
A. Clonidine	Drowsiness, fatigue, dry mouth, constipation, dizziness	Rebound hypertension, concomitant use of other CNS depressants
B. Guanethidine	Postural hypotension, diarrhea, weakness, nasal stuffiness, retrograde ejaculation, bradycardia	Heart failure, renal failure, severe bradycardia
C. Methyldopa	Drowsiness, fatigue, dizziness, dry mouth	Hepatic disease, hyperpyrexia, hemolytic anemia, (Coombs positive)
D. Metoprolol†	Bradycardia, anorexia, nausea, lightheadedness, insomnia, fatigue	Heart failure, asthma, diabetes, peripheral vascular disease
E. Nadolol†	See D.	See D.
F. Prazosin	Postural dizziness, palpitations, headache, drowsiness, weakness, nausea	Postural syncope (first dose)
G. Propranolol†	See D.	See D.
H. Rauwolfia alkaloids	Nasal congestion, depression lethargy	Mental depression, peptic ulcer disease
Vasodilators		
A. Hydralazine	Headache, tachycardia, palpitations, nausea	Coronary artery disease lupus-like syndrome (rare at recommended doses)
B. Minoxidil‡	Tachycardia, palpitations, edema, hypertrichosis, mastodynia	Coronary artery disease, fluid retention, congestive heart failure

All drugs listed in alphabtical order. This does not indicate preferential order of usage.

* Sexual dysfunction may be seen with any of the adrenergic inhibiting agents.

† Myocardial infarction and sudden death have been reported following sudden withdrawal of beta-blockers in patients with preexisting coronary disease. Cardio-selective beta-blockers (such as metoprolol) administered with caution in low dosage may be useful in hypertensive patients with asthma or peripheral vascular disease.

‡ Not generally recommended for uncomplicated hypertension. The risk/benefit ratio for this drug.is acceptable in patients with renal insufficiency.

The 1980 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure.

- Reinforce verbal instructions with material and memory aids. Family members or others involved in the therapy process can also receive material. Consider the patient's hearing and sight impairments, educational background, and present mental status when selecting these aids.
- Review medications for the first few minutes of each visit. Ask specific questions and listen for problems such as side effects, forgetting to take medication, poor eating habits. This is especially important when the stated treatment goal is not being achieved.
- As much as possible, simplify the number of medications and treatment schedules to encourage adherence. Encourage the patient to use a medication calendar or diary, to label drug containers clearly, and to destroy or return any and all medications not used.
- Prescribe new medications in small quantities to reassure the patient with limited financial resources. Occasionally drug companies will provide free drug samples which you can use to introduce a drug to a patient.
- Try to insure that patients acquire and refill medications. Nurses and pharmacy personnel may facilitate adherence. Spend a portion of one or more visits with the patient's spouse, a close relative or others, to build support for the total hypertensive control regimen.
- Encourage patients living alone to identify with a neighbor or friend who can offer personal reinforcement and support, and perhaps provide transportation for the office visit.
- Identify social agencies, health departments, or senior citizen service programs when appropriate. They can assist the elderly patient with special needs, including benefits, meals, nursing home visits, transportation, or other services. The cost of many of these services may be reimbursable by private insurance or public assistance programs. Programs that give the elderly "a chance to get out" can improve morale.

Summary

Hypertensive treatment of the elderly patient requires an approach that differs from that used in younger individuals. What has been provided is a presentation that involves basic philosophical concepts concerning the treatment of the geriatric patient with hypertension. These concepts include certain principles that will ensure effective control of hypertension in meeting established goals. The principles of evaluation and prescription in the total health care of the geriatric patient have been summarized.[8]

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Vox Dox

Vox Dox Editor:

The editorial comment on "The Trouble From Paradise" in the May 1982 issue of *The Journal of the Kansas Medical Society* discusses the concept of Original Sin.

As a reader of the works of Pelagius, I wonder if this editor is also a follower of his teachings? Pelagius was a British monk who lived in the fourth century, and his main contribution to theology was his denial of the doctrine of Original Sin. However, he was declared a heretic by none other than St. Augustine, and in spite of Pelagius' numerous followers at that time, the Christian religion maintained the theological concept of Original Sin.

My main comment on the recent legislative decision is that the theological, legal, and medical aspects of abortion, while closely interrelated especially in relation to an elective abortion, should be primarily viewed as a moral problem for woman and not as a social decision made by men.

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Senescent T Cell

Aging and the Immune System

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THE AGING PROCESS proceeds at different rates in each individual as well as affecting individuals differently. The immune system participates in unique ways as humans grow older. The thymus, most active at the time of sexual maturity, declines in both size and function subsequently. Its role in the differentiation of T-lymphocytes which in turn are necessary to regulate B-lymphocyte function may, as function diminishes, lead to impaired regulation of B-lymphocytes function, decreased antibody production, the production of autoantibodies, and in benign monoclonal gammopathy. Alterations in cyclic nucleotides and related enzymes in lymphocytes with aging also herald a decline in responses of lymphoid cells to immunological stimuli. There is a close relationship between aging and some quite small segments of chromosomal material which may be represented by the main histocompatibility complex (MHC) which controls many immune functions in humans. Aging and the immune system is much more thoroughly covered in several recent review articles.[1-3]

Careful anatomical studies done 50 years ago showed the mass of the human thymus gland, which is well maintained until sexual maturity, shrinks rapidly after that and by the age of 45 or 50 years the lymphoid mass is only 15 per cent of its maximal mass. The immunological function of the thymus, first recognized in the early 1960s, is now well established in cell-mediated immunity and in the regulation of the immune response. Lymphocytes arising in the bone marrow migrate to the thymus gland where differentiation into T-lymphocytes occurs.

This differentiation process is not completely understood, but it is known that the microenvironment of the gland, as well as hormones produced by the gland, are important in T-lymphocyte differentiation. A receptor for sheep erythrocytes, acquired in the thymus by the T-lymphocyte, causes mature T-lymphocytes to form "rosettes" with sheep erythrocytes. The percentage of thymic lymphocytes with this marker decrease with age from 85 per cent at age 20 years to 65 per cent at 50 years and 50 per cent at 80 years of age.[4] Even with the involution of the thymus, the number of peripheral T cells does not change significantly.

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Immune system functioning diminishes gradually with aging. Most striking is the involution of the thymus and its effect on the T- and B-lymphocytes. There is reduced antibody response to foreign antigens, but an increased response to self antigens (autoantibodies) and an increase in circulating immune complexes. Diseases of accelerated aging show early immune senescence. Increased longevity, at least in some animals, is associated with prolonged immunological vigor.

T cells appear to be extremely long-lived and replacement cells even from a limited amount of thymic tissue may be sufficient to replace the small number destroyed. T cells in lymphoid compartments other than the blood and possibly the spleen are not readily measurable and may be reduced in older individuals.

The thymus also plays a crucial role in the maturation of B-lymphocytes since exposure to T-lymphocytes is necessary before B-lymphocytes can respond to an antigen and produce immunoglobulin. In mice, T cells from older mice are severely impaired in their ability to mediate B cell maturation when compared to T cells from young mice. Several substances from the thymic gland — thymopoietin, thymosin, facteur thymique serique (FTS) — have been isolated, purified and amino acid sequence determined, and have been considered to be thymic hormones. The serum concentrations of each of these hormones has been shown to decline with age. Thymopoietin levels in human serum are constant between birth and age 30 years. There is then a steady decline to no activity at age 60 years.[5] FTS in human serum begins to fall after the age of 20 years

and cannot be detected after the age of 50 years.[6] The activity of thymosin alpha 1 seems to decline even earlier.

More than 50 years ago it was shown that antibody to the human erythrocyte A and B antigens declined with age. A few years later it was shown that the levels of human antibodies to sheep erythrocytes, the thymic dependent antigen, also declined with age, with highest levels in individuals 15-20 years of age. Response to a variety of foreign antigens is reduced in the elderly as compared to young individuals but there is an increased response to self antigens with age; however, no decline in the concentrations of serum immunoglobulin is demonstrable. Increase in autoantibodies and a decrease in antibody response to foreign antigens has also been observed in experimental animals with aging. In addition decreased antibody response, delayed cutaneous hypersensitivity, tumor and graft rejection, and resistance to mycobacterial, viral, and fungal infection are impaired in the elderly. Lymphocytes from older humans sensitive to PPD can be studied in vitro by culturing with the antigen. Such lymphocytes proliferate and incorporate labeled thymidine at rates less than lymphocytes from sensitive younger people under similar culture conditions. The proliferative response of T-lymphocytes from older humans stimulated by mitogens is also impaired, as is the capacity of these mitogen responsive T cells to divide repeatedly in culture. While the number of T cells dividing for the first time was comparable in cultures from old and young donors, the number of T cells from older donors that divided for the second or third time was only one-half or one-quarter, respectively, of the number of T cells from young donors. These findings indicate intrinsic defects in the function of T-lymphocytes of older persons. Studies with B cells and macrophages suggest that human macrophages, like human B-lymphocytes, are not significantly impaired with age. The predominant defect in immune function of lymphocytes from older humans appears to reside in the T-lymphocyte populations. With age, T cells have an impaired response to T cell stimuli and also suppress B cell responses.

Interesting changes have been demonstrated in the biochemistry of lymphocytes of young adult animals and humans as compared to older animals and humans. Lymphocytes of older humans and mice and in Down's syndrome (which is known to show many features of accelerated aging) show a five-fold decline in cAMP, a seven-fold increase in cGMP with a 50-fold decrease in the cAMP/cGMP ratio. The enzyme, adenylate cyclase was elevated about 3.5-fold while guanylate cyclase decreased three-fold.[7] These changes must represent intrinsic biochemical alterations in lymphoid cells and may have general significance as a fundamental age-related lesion, and be in part responsible for the immunodeficiency of aging.

The main histocompatibility complex (MHC) of genes is located on chromosome #6 in humans and #17 in mice. Genes within the MHC in man include those of the human leukocyte antigen (HLA) system containing five recognized histocompatibility loci, namely HLA-A, -B, -C, -D, and -DR. Within or close to the MHC are genes for coding for complement proteins, for certain disease susceptibilities, structural genes for red cell enzymes and antigens, various regulatory genes, and genes for different types of receptor and binding proteins. This area is implicated in T cell and B cell specific differentiation, and governs the ability to react against foreign or allogeneic material as well as self recognition. Response to corticosteroids, levels of testosterone and testosterone-binding protein, iron metabolism, and others are related to the MHC.

HLA antigens have been found in all human nucleated cells that have been examined as well as in saliva, serum, seminal fluid, and milk. The HLA-A and -B regions in humans are probably involved in recognition in cell-mediated lymphocytotoxicity (CML). The HLA-D antigen in humans serves as the stimulator factor required for recognition of non-self in mixed lymphocyte reactions (MLR).

Genetic analysis of inbred mice strains has been most helpful in evaluating the importance of the MHC in the genetic control of aging. Congenic mice, identical except for small segments in the major histocompatibility complex (H-2 system in mice) have different maximal life spans, and mice with the longest life spans have the longest preserved immune competence.[9] Thus genes within the MHC may not only modulate life span, but may also influence the rate of aging of the immune system.

Various studies attempting to correlate the HLA complex and aging in humans have not been too convincing. Man is an outbred species and many factors that may influence longevity are present. There is a decrease in the incidence of HLA-A1, -B8 in older females which may reflect the known correlation of the HLA-B8 antigen with auto immune susceptibility in humans. There are reports of an age-related increase in HLA-Bw40. In 122 humans over 90 years of age, a statistically significant increase in HLA-B4 and -B6 was found.

There are two known ways to extend life span in animals. One is the lowering of internal body

temperatures which is feasible only in poikilotherms. The other is caloric undernutrition initiated very early in life. Caloric undernutrition does lead to the retention of a vigorous immune response in older animals and a significant decrease in the incidence of autoantibodies and a delay in thymic involution. Gradual but severe caloric restriction beginning in adulthood in mice may lead to an immunological rejuvenation and possibly some life span extension. Thyroxin, various thymic extracts, levamisole, 2-mercaptoethanol, free radical inhibitors, synthetic polynucleotides, and coenzyme Q_{10} have been reported to ameliorate senescent changes in the immune system.

Human diseases with features of accelerated aging which have been studied immunologically include Down's syndrome, diabetes mellitus, several of the chromosomal instability syndromes, and systemic lupus erythematosis.[10] Down's syndrome patients demonstrate a decrease in life span, pathological changes characteristic of Alzheimer's disease in all subjects over 40 years of age, lesions in the microtubules and neurons characteristic of Alzheimer's disease, early involutional changes in the thymus (even in newborns), and morphologic features of aging such as pulmonary and endocardial fibrosis, diffuse degenerative changes in the endocrine glands, increased amount of lipofuchsin pigment, premature graying of hair, and decreased replicative capacity of fibroblasts in culture. Many investigators have documented evidence of immune senescence in Down's syndrome. There is a marked decrease in delayed skin sensitivity, thyroid antibodies are significantly increased, lymphocyte depletion and enlarged Hassell's corpuscles occur in the thymus, as well as the previously discussed changes in nucleotide biochemistry in lymphocytes that occur with aging. The immune functional changes in Down's syndrome are quite characteristic of what one would expect for a progeroid syndrome (accelerated aging).

Persons with insulin-dependent diabetes mellitus show an increased incidence of autoantibodies to gastric, thyroid, and nuclear antigens. In insulin-dependent diabetics less than 40 years of age, the incidence of autoantibodies equals that of a 60-year old normal population. These and other findings lend support to the fact that insulin-dependent diabetics show accelerated aging.

With the increase in survival time of patients with systemic lupus erythematosis (SLE), features consistent with accelerated aging have become apparent. These include loss of hair, skin changes, hypertension, osteoporosis, hypogonadism, diabetes mellitus or characteristic changes in the glucose tolerance curve, degenerative vascular disease including atherosclerosis, decreased pulmonary functions, increased susceptibility to one or more types of neoplasm and dementia, or related central nervous system pathology. SLE is strongly associated with HLA-DR3 and -DR4.

Chromosomal instability syndromes include Werner's syndrome, xeroderma pigmentosum, and ataxia telangiectasia. Werner's syndrome has clear-cut immunological abnormalities with autoimmune features and has been called adult progeria. There are also immune deficiencies in xeroderma pigmentosum and in ataxia telangiectasia.

Diseases of normal aging such as arteriosclerosis, renal disease, rheumatoid arthritis, benign monoclonal gammopathy, amyloidosis, and Alzheimer's disease have evidence of immune abnormalities but the importance of these abnormalities as an etiological factor has not been established. There is a significant association between Alzheimer's disease and HLA antigen-B7 and -Cw3 and particularly for the haplotype -B7Cw3 which is quite rare in the general population.

There is excellent evidence in animals that the genes of the major histocompatibility complex influence the rate of aging of the immune system as well as the maximal life span. There is suggestive evidence that this is also true in humans. Furthermore, the two means known to increase the maximal life spans of animals — caloric undernutrition and reduction in body temperature — are both associated with prolongation of immunological vigor. In humans there is a wide variation in immune competence after middle age, and preliminary evidence points to an association between immune competence and long life. There is an increased risk of death in older persons with autoantibodies, with low suppressor activity, or with greatly impaired cutaneous reactivity. Further study is needed to determine whether these relationships are causal.

An immunological theory of aging may or may not be valid; however, it is almost certain that immune senescence plays an important part in aging. It is related to the increased incidence of infection in the elderly and it is also likely that it contributes to the increased incidence of neoplastic disease among the elderly. The disordered state of self-tolerance, the formation of autoantibodies, and the circulation of immune complex may accelerate arteriosclerotic vascular disease and other diseases. As more is learned about these processes we may learn to influence aging and its diseases by immune therapy.

(Continued on page 392)

Nutrition and Aging

Assessing the Nutritional Status of the Elderly Patient

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ELDERLY, it has been said, is anything in excess of 15 years beyond one's own age. *Aging* was defined by Theodore Reiff, M.D. as a physical or chemical process, unrelated to time; *life* as the biological events taking place through time. It is true that people age gradually and after different numbers of years. Each person's rate of atrophy or entrophy depends on physical, emotional, psychological, and nutritional factors in his/her life.

In 1900, 4 per cent of the United States population was past 65 years of age. In 1975, the percentage had risen to 10.5 per cent, and continues to rise. What happens during aging will be increasingly relevant to more of the population.

The *American Journal of Medicine* quotes several surveys reporting that possibly 50 per cent of the elderly population consumes less than two-thirds of the recommended dietary allowances (RDAs) of calcium, iron, thiamin, riboflavin, niacin, vitamin A, ascorbic acid, and calories. Some of these studies did not include analyses for minerals such as zinc and magnesium. Adequate nurtriture of these two minerals is of growing concern to nutritionists.

Physiological Changes

A number of common physiological changes due to aging affect nutritional status. Decreased sensitivity to salt and sugar solutions has been documented in relationship to aging. The ability to differentiate between flavors also decreases. Food is a source of pleasure to most of the human race; when pleasure decreases, food choices change. The nature of the changes may be in personal choices of foods, or in reduced variety or quantities of food chosen. Decreasing the variety and amount of food eaten lessens the probability of consuming adequate nutrients.

Another change is the decrease in secretion of hydrochloric acid. This curtails the chemical changes of food in the stomach where food is prepared for further digestion. Altered pH also affects the activity of digestive enzymes, leaving food and nutrients less available to the body.

This, plus age-related changes in the gastrointestinal mucosa, decreases absorption of nutrients in the small intestine. Vitamins, minerals, and sources of calories remaining in the small and large intestine make less energy available at the cell level. The nutrients necessary for utilization of the energy that does arrive in the cell, are available in smaller quantities. This begins a cycle, for poor cellular nutrition decreases appetite, making adequate nutritional intake less probable. Cells in the gastrointestinal tract have a short life span, and their renewal and replacement will be quickly impaired without the raw materials to create new tissue. Absorption will thus be further decreased, and food more poorly utilized.

Many of the physiological and social changes related to the aging process affect the nutritional status of the elderly. Adequate nutrition can improve the quality of life, and physicians should help their elderly patients to maintain an appropriate nutrient intake.

Another common physiological change is that of decreased intestinal motility. This factor, unless dietary fiber and fluids are adequate, results in constipation and hemorrhoids, common in those of advancing age. Medications that slow motility also are a factor. Some nutritionists relate increased time for wastes to pass through the intestinal tract with an increased risk of cancer of the colon.

The proportion of lean body mass to body fat also changes as a natural consequence of aging. The fat tissue remaining has a lower rate of metabolism than the replaced muscle, so overall calorie needs, as well as physical strength and ability to exercise, decrease with this change. Cellular needs for other nutrients remain at the same levels as in earlier life. When calorie needs and intake decrease, foods must be chosen with more care and be of high nutrient density, or the amount of nutrients consumed may be inadequate. Any diet routine of fewer than 1000

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calories/day is most likely to be inadequate in vitamins and minerals, no matter what foods are chosen. People who consume more than 3000 calories/day are likely to have an adequate intake. Between that range — which covers most people — it is the quality of specific foods chosen that determines whether the diet is nutritionally adequate. Keeping an optimum proportion of calories from protein (15%), carbohydrate (50%) and fat (35%), and exercising sufficiently helps to slow the process of change from muscle to fat tissue. Perhaps the aging of the current population of joggers and fitness buffs will confirm this.

The immune system in the elderly is generally-considered to become less efficient, resulting in an increased incidence of infection and longer healing times. Nutrients such as protein, ascorbic acid, and zinc play especially vital roles in the body's overall ability to fight infection and heal itself. Broken bones require adequate calcium, vitamin D, ascorbic acid, protein, and magnesium to heal efficiently.

Aluminum toxicity is being cited as a cause of senility, possibly that being observed in the elderly. Food-related sources are baking powder and the cooking of acid foods in aluminum pans.

Chromium is known to decrease in concentration in the body tissues throughout the life span. The need for chromium is said to increase in old age and to be associated with glucose intolerance, elevated serum cholesterol, the presence of sclerotic plaque, and other health problems.

The abnormal changes seen with diseases associated with aging are related to nutrition status — either as cause or effect or both. Over-nutrition (obesity) increases the likelihood of diabetes, heart disease, and hypertension. The underweight person may have little reserve of protein or stored energy (fat) to fight disease or to maintain life following injury or accident. Nutritional factors are being investigated as the cause and treatment of some cancers. The National Cancer Institute will be funding investigations into the use of water-soluble vitamin A in the treatment of cancer plus other vitamin/cancer relationships.

Social Changes

Common life circumstances frequently change an individual's eating habits sufficiently to reduce the adequacy of nutritional intake. Eating is often a social act, and when an elderly person eats alone, either at home or at restaurants, s/he may tend to eat less. Some foods, such as stews and casseroles, are difficult to prepare in one-serving amounts. Sometimes people do not go to the trouble of fixing food for only themselves. Nibbling or snacking may take the place of meals with a corollary change in types of foods eaten. Frequently these changes include an increase in fat, salt and sugar, and a decrease in fresh meats, fruits, and vegetables with their nutrients.

An estimated 50 per cent of people over age 65 years have no natural teeth. Eating without teeth or with dentures changes many people's food choices — generally resulting in lowered consumption of meats, fresh fruits and vegetables, along with the vitamins and minerals they contain.

Studies reported in the *Journal of the American Dietetic Association* indicate income and education are important factors in predicting the adequacy of a senior citizen's diet. Low income means less food for many — commonly less fresh foods.

Another factor affecting food choices in the elderly is decreased mobility and agility. This can involve the ability to get to the grocery store to shop, or to stand in the kitchen with a cane trying to manipulate a can opener with arthritic fingers.

The difficulty of changing established habits may be either a positive or negative factor when those habits involve food and reach back 65 years or more. Many of the older generation have habits that contribute to good health. They eat breakfast, eat larger meals at noon rather than before bedtime, and may prefer food such as oatmeal to sugar-coated puffs. Nutrition may be quite a challenge to future health care providers when the present fast-food-twinkie-coke generation reaches the elderly stage.

Patient Nutrition Histories

A physician with limited time and a concern for patients' nutritional status might ask, "What questions would yield the most information about probable nutritional health in the least time?" Following are some suggestions for eliciting a general picture of a patient's eating habits accompanied by some considerations for evaluating the answers:

1. Do you eat alone more than one meal a day?

A "yes" answer would call for further inquiry into what is eaten and when. Some situations may prompt a referral to a "Meals on Wheels" or congregate eating program. Most participants in these programs have been shown to have a better nutrient intake than people in similar situations who do not participate.

2. Do your teeth or dentures cause you to omit many foods you used to eat? Which ones?

If numerous foods or whole categories of foods are omitted, there is often a low intake of specific nutrients. For example, meat is the primary source of iron for many people; omission of milk and cheese

generally leads to a low intake of calcium and riboflavin; leaving out grains and cereal products can create a low intake of thiamin.

3. Do you often eat in fast food restaurants?

This commonly leads to a high sodium, high fat, low vitamin A, and low fiber intake. Does the patient allow for this in other meals?

4. Are you troubled with constipation?

What is the patient's intake of whole grain bread, cereal, crackers, and similar foods? Is s/he eating bran for a supplement but failing to adequately increase fluid intake?

5. Do you take vitamin, mineral, protein, or other supplements? Which ones? What level?

Many people believe their supplement is complete when, in fact, it is not. They therefore are victims of a false sense of safety. This is particularly true of calcium; it is included in many vitamin/mineral supplements in very low amounts. Toxic levels of vitamin A and D are available. Too much vitamin E (more than 400 IU/day) has been shown to increase the probability of thrombophlebitis in some people. A vitamin/mineral supplement is advisable for many elderly, but the content should be chosen to conform to nutrient requirements.

6. How many calories per day are in the form of ethanol and sucrose?

It is not uncommon for individuals to consume 25-35 per cent of their calories from these nutrient-poor calorie sources. Unless food is especially well chosen, it is unlikely to compensate for these "empty calories" and maintain the intake level of vitamins and minerals necessary for the enzyme systems that metabolize these calories.

7. Do you eat 5-6 ounces of meat or substitute daily? Four servings of fruits or vegetables daily? Four servings of a grain food daily? Two servings of a dairy product daily?

These *do not guarantee* meeting the Recommended Dietary Allowances for nutrients, but *without* this assortment of foods, it is very likely that the RDAs will *not* be met.

The two most common nutrient deficiencies in the diets of the elderly are those of calcium and vitamin A. Increased intake of calcium to 1200-1500 mg/day (compared to the RDA of 800 mg/day) has been shown to reverse negative calcium balance and halt the progress of osteoporosis.

An alternate to nutrition history-taking by the physician is a complete nutrition history and assessment by a Registered Dietitian, possibly supplemented by a computerized diet analysis. Dietitians in private practice (a new and growing resource) and those employed by physician practice groups make this type of nutritional care available outside of the hospital setting.

Physical Signs

Suboptimal nutrition, as opposed to severe malnutrition, is likely to appear in the form of generalized difficult-to-pinpoint symptoms. For any age group, having half enough nutrients will not always result in an identifiable disease state, but will not produce an optimally functioning body or mind either. A wide variety of physical abnormalities can result from less than adequate levels of nutrition.[1]

Summary

Numerous social and physiological factors contribute to suboptimal nutrition or frank malnutrition in the elderly. Astute history taking and observation of physical signs with the possibility of nutritional causes in mind can prevent poor nutrition from causing significantly impaired health or contributing to existing health problems. Optimal nutritional health can improve the quality of life for the elderly.

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Medical
ETHICS

After Quinlan

Guidelines and the Ethics of Dying

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It is a recurring and melancholy fact of history that individuals, on occasion, have made a greater mark on their time in the act of dying than they ever could have in living. Sacco and Vanzetti, Joan of Arc, and for that matter, Jesus Christ, are in that category. A more recent example is that of Karen Ann Quinlan. Her dying process, which is still going on, almost certainly will be seen by future historians as a watershed in the application of technology to the act of dying. Before Quinlan there was little public dialogue on the right to die or the definition of death. Since then the dialogue has spread to involve jurists, theologians, philosophers, ethicists, and the public at large.

It has now been some seven years since Karen Ann slipped into coma. During that time the meaning of Quinlan has been debated by a great many good minds. One of the more important outcomes is that the essential question remains unresolved, and indeed, if anything, is more problematic than ever. Which is not to say no progress has been made. On the contrary much public good has derived from Ms Quinlan's private misfortune. The public has insisted on a greater voice in determining the nature of medical intervention in the dying process. Physicians have been forced to examine their tendency to use life-sustaining technology simply because it exists. Jurists and legislators have tried, with varying success, to define the circumstances under which life-prolonging technologies may be discontinued without legal jeopardy to the medical attendants or survivors. Due in part to Quinlan, the entire subject of death and dying has been raised in the public's consciousness, and death has become a subject of serious discussion rather than a national neurosis. And yet, the basic issue that made Ms Quinlan's situation different from its predecessors remains: namely, when there is dispute between the most interested parties — in this case the parents and the physicians — who shall decide whether to remove life-sustaining technology?

Much public good has derived from the private misfortune of Karen Ann Quinlan. Principally this good relates to the dialogue precipitated between health professionals and others. Yet despite all of the discussion of Quinlan and important similar cases, no single set of guidelines — medical, ethical, or legal — can be constructed to direct physicians in all cases in which the issue is prolonging life.

The Quinlan case began on April 14, 1975, amid circumstances that have never been completely clarified. According to her friends, Ms Quinlan had been depressed and taking tranquilizers, and while drinking gin and tonic she started, in their words, "nodding out." As they took her upstairs, they

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noticed she had passed out, and "was turning blue." For the next 30 minutes or so she was given mouth-to-mouth respiration — 15 minutes by friends and 15 minutes by a policeman. At this time she was turned over to the Lakeland emergency squad. It is believed that she never developed a pulse or respirations during the period before arrival at the hospital. In the emergency room her pulse and skin color returned, and she began breathing intermittently. On physical examination her pupils reacted, and she showed changes in her posture when stimulated which suggested that her cerebral cortex was damaged. Later, the electroencephalogram showed electrical activity, although diminished.

Because she could not trigger the respirator, she was placed on a breathing machine where she remained during the legal chapters of her story, which occupied the next year or so. Initially she was in a state of "sleep-like unresponsiveness," but with the passage of time she developed "awake unresponsiveness," in which cycles of sleep alternated with periods in which her eyes were open but did not "see" in the sense of following the actions of nearby persons. She settled into a "persistent vegetative state," meaning she had spontaneous regulation of her pulse and blood pressure, and to a lesser, but at the time unknown degree, her respirations. Over the months her weight dropped from 120 to 80 pounds, and she developed severe contractures in the fetal position.

At the outset, Karen Ann's parents believed she would recover, and so they authorized all life-supportive measures. Gradually it became apparent that she would not recover, and finally her parents signed a release, freeing the medical attendants and hospital from any legal liability that might result from removing her from the breathing machine. However, her principal physician, Dr. Robert Morse, refused to remove the respirator, and the impasse developed that made Quinlan's dying a media event.[1] On September 10, 1975, Mr. Quinlan petitioned the Superior Court of New Jersey to have himself appointed Karen's guardian for the express purpose of authorizing her removal from the respirator. The presiding judge, Robert Muir, Jr., ruled against Mr. Quinlan, and appointed an unrelated guardian to oversee Karen's interests. Physicians following the case must have heaved a collective sigh of relief at that point because Judge Muir stated explicitly that decisions about medical care were the physician's resonsibility — *i.e.* medical, not legal, in nature. In his words, "The morality and conscience of our society places this responsibility in the hands of the physician. What justification is there to remove it from control of the medical profession and place it in the hands of the courts?"[2]

But as everyone knows, that was not the last judicial word in the matter. The parents appealed to the New Jersey Supreme Court, which on March 31, 1976, reversed the lower court, saying that no interest of the State could compel Karen to remain alive in the face of overwhelming evidence that she would never emerge "from her present comatose condition to a cognitive sapient state."[3] The Court appointed Mr. Quinlan guardian, and Karen was removed from the respirator. She did not die, however; she began breathing on her own and now resides in a nursing home.

The Quinlan case could serve as a textbook for a course covering the entire range of ethical issues surrounding dying and the definition of death. The present essay is limited instead to the proposition that no single set of guidelines — moral, legal, or medical — can be fashioned to resolve the infinite range of ethical considerations inherent in the physiology of dying, and that the attempt to derive such systems rather uniformly disserves patients and physicians alike. In the Quinlan case a number of approaches were attempted, and it appears that every individual in the matter acted in perfectly good faith, and only after genuine soul-searching. Yet it is probably equally true that none of the principals is pleased with the outcome.

Karen Ann's physician relied on medical criteria when he decided Karen's brain was not dead, and that he could not shut off the respirator. If we take just one of the several sets of criteria medical people have devised to cover this circumstance, we find irreversible coma defined as: (1) unreceptivity and unresponsitivity; (2) no movements or breathing; (3) no reflexes; and (4) flat electroencephalogram.[4]

Strictly speaking, Karen did not satisfy a single one of these criteria, which is to say, by general medical agreement her brain was not dead. Thus, rather than being dogmatic or callous, her physician was merely following the best medical guidelines available to him at the time.

Faced with the implications of other Karen Quinlans — that is, patients who do not satisfy scientific criteria of brain death, but who are in a vegetative state from which recovery is exceedingly unlikely — health professionals turned to other disciplines for assistance, specifically philosophy and religion, two pursuits that have been occupied with questions of human values over the centuries. One promising lead was the concept of personhood which says in effect that vegetative life alone does not make a human being a person, but that certain other charac-

teristics are necessary as well. These include cerebration, self-awareness, ability to communicate, purposiveness, self-control, memory, sense of time or futurity, and a desire to live. The personhood idea led to a good deal of fruitful thought and dialogue, but it suffers several drawbacks that limit its usefulness on the front line of medicine. For example, as nearly as they can be determined, the qualities that comprise personhood are absent in newborn infants. Even though the normal neonate lacks most of the requisite qualities, few would want to deny it personhood. Second, how many of the qualities must be absent and to what degree, before personhood can be said to be absent? Certainly the individual with total amnesia is not to be denied personhood because memory alone is missing. The concept, if it is to help in cases such as Karen's, must be sufficiently precise to indicate within rather narrow limits, just when a person becomes a non-person. This is because removing a respirator is a precisely timed and highly unambiguous act. A final objection to the personhood concept is that we do not have the technology to measure precisely such qualities as self-awareness, ability to communicate, and desire to live. Are we to say a fetus has no desire to live simply because it cannot articulate such a desire at a time when every embryological event crowded into its gestation is preparing it to live above all else? According to one report, Karen's mother has determined from Karen's eye movements that her favorite piece of music is Judy Collins singing "Amazing Grace." Whether Karen has communicated this information to her mother depends ultimately on one's definition of communication.

Unfortunately, then, for those having to make the initial decisions in these matters, personhood and related concepts, such as potential for life and quality of life, are useful only in extreme cases — the A-Z cases, they have been called — and lose their practical utility in the gray zone — the M-N cases such as Karen's.

The ethical approach fails in a global sense on further grounds. The vexing fact is that there is no uniform basis for medical ethics, a conclusion that most people involved in these matters have not yet fully acknowledged.[5] Those who would advocate Christian ethics as a basis for making moral judgments have the Bible as a point of agreement or disagreement. Similarly, if one chooses utilitarian ethics as a determinant of ethical decisions, one has a body of rational discussion that has accumulated to utilitarianism over the years. But no such basis exists for medical ethics. If you are an atheist or an agnostic, or Muslim or Jew, you have no overriding reason to accept the Christian premises for ethical decision making in a given dying situation. This is a major reason why there has been no substantial resolution of the abortion issue on the national level since the Supreme Court imagined it had settled the matter in 1973. The contending parties in the abortion debate simply arrive carrying basically different premises. If you believe in life-at-conception, there is little chance for accord with someone who believes in life-at-birth. And what is more important, *there is no rational basis for accepting one premise over the other*. The same applies in Quinlan. Whether you choose to label Karen a person or not depends on the values you choose in defining personhood, and these values cannot be validated by either medical science or rationality.

When it developed that Quinlan cases could not always be resolved on medical grounds, and that the offerings of other disciplines traditionally interested in values such as philosophy and theology were similarly wanting in final answers, America — as it has too often lately — turned to the courts. Therein too, no panacea was discovered, as most thoughtful biologists would have predicted. As indicated, Judge Muir saw the Quinlan case as medical, not legal. When the New Jersey Supreme Court overruled Muir's opinion, however, they said in effect, any time there is a dispute in these matters, the court may be a proper avenue for resolution.

During the next few years, the matter did not remain untouched by the law for long. With Quinlan, the court had indicated that there was a certain class of patients who could be treated differently from others, even to the extent of withdrawing life-support systems. These were individuals who were in a vegetative state and had no realistic possibility of returning to any semblance of cognitive or sapient life. The next prominent case involved Joseph Saikewicz of Massachusetts, who was a 67-year-old mentally retarded man with an IQ estimated at about 10 and a mental age of 3 years. In 1976 Mr. Saikewicz developed acute leukemia, and those in attendance petitioned the court to withhold treatment. The court acquiesced. Speaking for a unanimous bench, the spokesman said that the probate court had full and sole authority to make such determinations.[6] This decision promised great difficulties for health professionals. If the courts have sole authority to withhold treatment in retardates, on a case by case basis, why not in all those who cannot finally speak for themselves, *i.e.* all unretarded but unconscious adults and all children as well?

Before the full import of Saikewicz could be realized, along came the case of Brother Fox in New

York. At age 83 years, while undergoing surgery for an inguinal hernia, Brother Fox, a member of the Catholic Order of the Society of Mary, suffered a heart attack which produced substantial brain damage followed by coma and the need for artificial respiration. Again the neurological verdict was one of hopeless coma. Father Philip Eichner, a long-standing friend, attempted to have himself appointed guardian, as Karen's father had, with the aim of discontinuing the respirator. The trial court granted Father Eichner's request, but the District Attorney appealed.[7] The Appelate Division of the New York State Supreme Court sustained the lower court judgment, but then set up a procedure for handling all such cases in the future.

In brief, although it is far from brief itself, the procedure was as follows:

1. The attending physician certifies that the patient is terminally ill and in irreversible coma;
2. The responsible person, *e.g.*, a close relative, presents the certificate to a hospital prognosis committee made up of no fewer than three physicians practicing specialties related to the patient's condition;
3. The committee confirms or rejects the certificate by majority vote;
4. If the prognosis is confirmed, the person(s) seeking it may go to court to try to become the guardian;
5. At that point the Attorney General and appropriate District Attorney must be notified and given a chance to have their own physician examine the patient;
6. Next, a temporary guardian is appointed to protect the patient's interests;
7. The court then determines that the prognosis is accurate by ''clear and convincing'' evidence and that the patient would have discontinued life support measures had he been able to make such a decision; and
8. The appropriate order shall be issued relieving all participants of civil or criminal liability.[8]

The response of health professionals to all this should have been one of metastatic horror. If nothing else there is the element of time. All manner of objections, motions, and other legal maneuvers could occur at several stages of the procedure, but the dying process usually does not respect such judicial niceties. Indeed Brother Fox died before the Appeals Court could even begin considering the case, and Saikewicz had been dead 14 months before the Massachusetts Supreme Court handed down the decision just mentioned.

This is the same sort of ignorance of biological imperatives that the U. S. Supreme Court exhibited in the 1973 abortion decision. In that instance the Court ruled that the states could pass laws regulating and even forbidding abortion during the last trimester of pregnancy. Even if physicians could always decide whether a fetus was in the second or third trimester, which they cannot, the situation is biologically ridiculous. Suppose a woman was three days short of entering the third trimester in a state that has enacted a law prohibiting abortion in the last trimester, when her husband sought an injunction on grounds that she was already in the third trimester. By the time the court could schedule and hold a hearing, the necessary physicians make the necessary examinations, and all this be reported back to the court, the infant would have been christened, if not weaned.

To the general relief of ethicists and health professionals alike, the story of Brother Fox had one more chapter. On March 31, 1981, New York's highest court, the Court of Appeals, struck down the clumsy and expensive procedures formulated by the Appellate Division of the Supreme Court (medical and legal costs of Brother Fox's case exceeded $100,000 before his heart gave out).[9] Despite its reasonable outcome, the Fox case will remain instructive to an anxious medical profession, because in it we saw how unwieldy and ultimately unworkable the legal mechanism would have to be in practice.

The legal story next involved the case of John Storar, a resident of a state facility in New York who was 52 years old, but had a mental age of about 18 months. In 1979 he was diagnosed as having bladder cancer, and his mother was appointed guardian to oversee his radiation therapy. In March, 1980, he began hemorrhaging from the bladder, and it was discovered that the cancer had metastasized widely. At that point the mother consented to blood transfusions, but in June 1980 she withdrew her consent because Mr. Storar fought the transfusions and was having increasing pain in passing blood clots. Administrators of the institution petitioned the court to continue the transfusions. The lower court backed the mother's position, but the New York Court of Appeals reversed the decision on grounds that there was no way to determine what Mr. Storar, himself, would want done. This was a problem, you may recall, which apparently posed no difficulty for the Court in the case of Brother Fox. In the opinion of at least one expert, the Appeals decision carried no legal analysis worthy of the term.[10] In effect they declared Storar an infant and likened his case to those of children of Jehovah's Witnesses, wherein courts have consistently held that parents may not

withhold life-saving medical treatment. Physicians may have some difficulty equating transfusions in terminally ill John Storar and an otherwise healthy child of Jehovah's Witness parents. As it stands now in New York "minors and that vast majority of the population" who become comatose without having executed a Living Will or expressed their feelings on the subject in front of reputable witnesses must undergo "all treatment for a condition which threatens [their] life."[9] In Storar it appears clear that the judge did not understand the medical facts involved, yet he made the case the basis for a sweeping conclusion that, if followed in practice, is bound to produce protracted suffering in many cases in which physicians and families agree that continuing treatment more nearly resembles human torture than humane caring.

In the case of Karen Ann Quinlan and those cases that followed, three solutions were tested and found ultimately deficient, each for its own reasons. Medical criteria for brain death simply cannot be constructed in such a way that the infinite variations of the disease process will not confound them sooner or later. Karen Ann did not suffer brain death according to medical criteria, but the chances are practically nil that she will ever become a functioning being. There are no ethical absolutes to be gained from the Quinlan case because it will never occur again. Saikewicz was not the same as Brother Fox because Saikewicz was mentally retarded. Nor from a moral standpoint would Saikewicz have been Saikewicz if his I.Q. had been 70 instead of 10. Quinlan and Fox were not the same as Saikewicz because they did not have terminal illnesses and he did. If Ms Quinlan developed acute monocytic leukemia tomorrow, would the court order the usual round of chemotherapy for her? No one knows, because there is no precise legal or moral precedent for such an eventuality. But there is no doubt that leukemia in Quinlan would change her situation, and as a result of this new biological situation, the ethical circumstances would change as well.

Definitions of personhood fail because there can be no agreement on which qualities are necessary and to what degree, and because the qualities defy quantification — and indeed, at times, defy all attempts to determine if they are present at all. Further, we lack the moral consensus necessary to define personhood in our system of ethical pluralism.

For its part, the law relies on precedent, a word that means little in the morality of dying. The law needs to hand down opinions that will direct all such future cases, and even increase the chances that they will not come to trial because they have already been decided and affirmed on past occasions. But once again the dying process is not susceptible to this treatment because each process differs. When a man holds up a bank, he can be dealt with in precise legal terms. The judge may find extenuating circumstances which will alter the sentence from case to case, but the basic determination of whether the man's action constituted bank robbery is amenable to legal definition. Not so with such concepts as personhood, potential for life, and quality of life.

Medicine's plea then should be simply for humility and restraint. The issues we are discussing are new. Our ability to sustain comatose persons on artificial respiration for prolonged periods is only some 15-20 years old. It should not surprise us that an exploding technology has showered us with terribly complex issues which were not foreseen in the Bible, the Hippocratic Oath, or British Common Law.

What is called for is medical, ethical, and judicial restraint. Physicians for their part must be willing to reexamine traditional professional values which allowed them to subscribe to a philosophy of life-at-any-cost at a time when there was little they could do to prolong life in any event. Ethicists should be willing to admit that there is no universally accepted basis for medical ethics, and that thus there is a limit to the contribution their discipline can make, at least at this time, and probably in the future as well. Above all we can hope for judicial restraint. For the moment the courts should refuse to engage any issue of medical morality that is not literally forced upon them, rather than reaching out for universal mechanisms to resolve biological complications and new technology that no one can predict today. When they must act, they should expressly limit their judgment to the case at hand, thus acknowledging that an identical case will never again appear before the bar. Physicians have an important contribution to make in this particular regard. They must make every effort to reach accord between their medical judgment and the wishes of the patient and the family. Had Dr. Morse been the Quinlan family physician, there is a possibility, if not probability, that her situation would have been resolved quietly and to everyone's best interest. If a physician opposes the Living Will concept in principle (as too many do without having analyzed the matter sufficiently) he must discuss the use of life-prolonging technology openly with patient and family, and strive for the consensus that will obviate any need for judicial intervention.

(Continued on page 340)



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Lyme Disease — "What's New in Old Lyme?"

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In late 1975, state public health officials discovered an unusual cluster of epidemic inflammatory arthritis in the inhabitants of the area surrounding Lyme, Connecticut — a small, semi-rural community adjoining Long Island Sound. Subsequent investigations by Steere's group at Yale School of Medicine have shown that "Lyme arthritis" (as it was initially called) is actually only one component of a disease complex that also includes a unique skin rash (erythema chronicum migrans), myocarditis, and meningopolyneuritis *(Table I)*. Evidence supports transmission by an arthropod vector, but the actual infectious agent has eluded detection. Because of its multi-systemic nature, "Lyme disease" has become the preferred term for this previously unrecognized disease entity.

Investigators have long sought an infectious etiology for common forms of inflammatory arthritis, including rheumatoid disease, Reiter's syndrome, ankylosing spondylitis, and systemic lupus erythematosus. Lyme disease must now be added to this list. The epidemiologic data pointing to an infectious cause for this disease is convincing, in spite of the fact that no evidence for infection by any known bacteriologic, viral, or fungal agent has been found. A recent report by Burgdorfer and co-workers (Science 216:1317-1319, 1982) detailed the isolation of a treponema-like spirochete from adult Ixodes dammini ticks, as well as presence of antibodies to this agent in the sera of human patients with Lyme disease. This discrepancy has led to the postulate that, in many rheumatic disorders, an infectious agent may trigger a systemic inflammatory response in the absence of overt infection.

There are two compelling reasons for physicians to become familiar with Lyme disease. First, early recognition and treatment may minimize subsequent manifestations associated with significant morbidity. Second, this newly described disease entity may serve as a pathogenetic model for other rheumatic disorders of unknown cause.

Epidemiology

Geographic and seasonal clustering of Lyme disease, with a peak incidence in summer and early fall, strongly implicates an arthropod vector. Field studies carried out after the initial recognition of Lyme arthritis revealed a new species of the Ixodes ricinus tick family *(I. Dammini)* clustered in the same geographical areas as outbreaks of Lyme arthritis. Currently, the known areas of Ixodes ricinus tick distribution in North America are northeastern United States, coastal areas of California and Oregon, central Minnesota, northwestern Wisconsin, northwestern Utah, and parts of Ontario, Canada. These should be considered endemic areas for Lyme disease, although case reports from Utah and Ontario

TABLE I
MAJOR FEATURES OF LYME DISEASE

Erythema chronicum migrans
Mono- or oligoarticular arthritis
Myocarditis
Meningopolyneuritis

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TABLE II
NON-SPECIFIC FEATURES OF LYME DISEASE*

Symptoms	*Signs*
Malaise and fatigue	Fever
Chills and sweats	Lymphadenopathy and splenomegaly
Headache, stiff neck	
Myalgias	Maculopapular rash
Nausea and vomiting	Pharyngitis
Abdominal pain	
Sore throat	

* Steere, A. C. *et al.:* Lyme Arthritis. *Arthritis Rheum.* 20:7-17, 1977.

TABLE III
DISTRIBUTION OF JOINT INVOLVEMENT IN LYME ARTHRITIS

Joint	*Frequency (%) (Patients with arthritis in specific joints)*
Knee	77
Shoulder	24
Elbow	19
Temporomandibular	17
Ankle, wrist, hip, small joints of hands and feet	9-13

are lacking. A transmissible infectious agent presumably exists, but exhaustive microbiologic and serologic studies have failed to provide a clue to the identity of the causative agent. Lyme disease affects children and adults without predilection for age, sex, or race.

Clinical Features

Erythema chronicum migrans: The skin rash of Lyme disease is striking, occurring in approximately 85 per cent of patients with the disease. It begins as a red macule or papule that rapidly expands with central clearing to form an irregular, annular lesion with a bright red border, which is sometimes indurated. There is occasional local pruritis, burning, or dysesthesia. The lesion may expand to 50 cm or more in diameter, but is self-limited and fades slowly over a median period of three weeks without scarring or pigmentary changes. The rash most often appears on the thigh, trunk, or buttocks. At least one-third of patients recall an antecedent tick bite in the area of the rash. A secondary crop of smaller, evanescent, annular lesions may develop elsewhere on the body during ensuing weeks. There is no mucosal involvement, and the appearance of the skin lesions on biopsy is non-specific.

Interestingly, the rash of Lyme disease is identical to the entity known as erythema chronicum migrans (ECM), an arthropod-borne disease well known in Europe, Scandinavia and Russia, which responds readily to oral antibiotic therapy.

A variety of non-specific symptoms and signs *(Table II)* frequently accompanies the appearance of the rash, as well as the other manifestations of Lyme disease.

Arthritis: Almost half of all patients with Lyme disease develop acute, asymmetric, inflammatory mono- or oligoarticular arthritis, usually affecting the knee and shoulder. Smaller joints may also be involved, albeit much less frequently *(Table III)*. As a rule, fewer than four joints are affected at any one time, although some patients have a migratory form, involving a total of up to ten joints. Twenty-five per cent of patients with arthritis have antecedent ECM. The timing of the onset of the arthritis is variable, but it develops in an average of four weeks (range 1-24 weeks) after the appearance of ECM. The remaining 75 per cent of patients without ECM may or may not also have cardiac or central nervous system (CNS) involvement, either singly or in combination. Attacks of arthritis tend to recur. The duration and frequency of subsequent episodes is highly variable. However, there is a general trend toward diminishing disease activity and resolution during a period of months to years. There is no difference in the frequency, duration, or interval between attacks of arthritis in patients with or without antecedent ECM.

Lyme arthritis is not always self-limited. At least ten patients have developed chronic inflammatory arthritis of one or both knees with marked synovial hypertrophy. Bony erosions were present in one case. Histologically the synovitis of these patients resembles the changes seen in early rheumatoid arthritis. Synovectomy has been remittive when it has been attempted.

An unusually high proportion of the patients with chronic arthritis have the leukocyte alloantigen, HLA-DRw2, which suggests a genetic predisposition for expression of chronic Lyme arthritis. This finding is reminiscent of the allelic associations observed in other rheumatic disorders such as anky-

TABLE IV
NEUROLOGIC FEATURES OF LYME DISEASE*

Symptoms	*Signs*
Headache, stiff neck	Cranial nerve palsies
Photophobia	Sensory loss
Sensory disturbance	Limb weakness
Somnolence, lethargy	Chorea
Emotional lability	Decreased hearing
Impaired memory and concentration	Babinski Sign
Visual disturbance	Cerebellar ataxia
Dizziness	Meningeal signs
Clumsiness	

* Reik, L. *et al.*: Neurologic Abnormalities of Lyme arthritis. *Medicine* 58:281-294, 1979.

losing spondylitis or Reiters syndrome (HLA-B27), and rheumatoid arthritis (HLA-DRw4).

Myocarditis: To date, the myocardial involvement in Lyme disease has been limited to the conduction system. Fluctuating atrioventricular blocks of all types are seen in approximately 8 per cent of patients with Lyme disease, occurring one to six weeks after the onset of skin or joint manifestations. Temporary pacing has ocasionally been used to alleviate symptoms, but oral prednisone (40-60 mg/day) usually results in the resolution of heart block in 12-24 hours. Intermittent episodes of first- and second-degree atrioventricular block may recur for months after the acute episode. No cases of permanent cardiac damage have been reported.

Meningopolyneuritis: Nearly 11 per cent of patients with Lyme disease have experienced neurologic changes *(Table IV)*. An impressive spectrum of specific lesions occurs. All are manifestations of widespread involvement of the central nervous system and its coverings. Aseptic meningitis, encephalitis, chorea, cerebellar ataxia, cranial neuritis (especially Bell's palsy), motor and sensory radiculoneuritis, mononeuritis multiplex, and myelitis have all been reported. Of these, fluctuating meningoencephalitis with superimposed cranial or peripheral neuropathy is the most frequent manifestation. Simultaneous constitutional complaints of fatigue, chills, sweats, arthralgias, backache, myalgias, and sore throat are expected. Common extraneural findings include fever, malar and maculopapular rash, as well as regional and generalized lymphadenopathy.

The time course of neurologic involvement in Lyme disease is highly variable. In a series of 18 patients, the average time of onset of neurologic abnormalities was seven weeks after the onset of ECM, but the range was 0.5-10 weeks. In ten patients, the abnormalities fluctuated for two to eight months before complete resolution, but the remaining eight patients continued to have abnormal neurologic symptoms beyond eight months.

Meningeal involvement responds to oral prednisone therapy (40-60 mg/day), with complete resolution usually occurring in 7-14 days. Other neurologic lesions do not improve with corticosteroid therapy, although some authors feel the onset of new lesions may be retarded by its administration.

Treatment

The fact that the European form of ECM responds to conventional antibiotic therapy prompted antibiotic trials in Lyme disease. Limited studies have demonstrated that only penicillin or tetracycline, given orally in conventioinal dosages for seven to ten days, are effective. With antibiotic therapy there is a clear reduction in time for resolution of ECM, total duration of joint involvement, and the likelihood of developing arthritis after ECM. Antibiotics afford no protection against cardiac or neurologic involvement.

As previously mentioned, oral prednisone therapy is indicated for Lyme disease patients with cardiac or meningeal involvement. Salicylates, non-steroidal anti-inflammatory drugs, or intra-articular corticosteroids have had variable and generally unimpressive effects on the manifestations of Lyme disease.

Laboratory Findings

The hematologic profile in Lyme disease is generally unremarkable, although the sedimentation rate and acute phase reactants may be modestly elevated. Serologic assays for antinuclear antibodies and rheumatoid factor are consistently negative, unless a concomitant autoimmune disorder exists. Serum complement levels are generally normal or increased during active ECM. Low complement levels have been observed in patients with high levels of disease activity and multi-systemic involvement.

Synovial fluid from affected joints is inflammatory, with elevated protein content and cellular infiltrate consisting mainly of polymorphonuclear leukocytes. Joint fluid cell counts average 10,000-25,000/μl, but may range from 500-100,000/μl. Complement levels in joint fluid averaged one-half the levels in serum.

Cerebrospinal fluid findings are non-specific. In patients with meningoencephalitis there is usually a lymphocytic pleocytosis. The cerebrospinal fluid

protein may be elevated and the glucose lowered, but not predictably.

Cryoglobulins are present in the joint fluid of all patients with Lyme arthritis; and approximately 50-75 per cent of patients with any manifestations of Lyme disease develop circulating cryoglobulins. Levels of circulating cryoglobulins tend to rise and fall along with the level of disease activity. The presence of cryoglobulins containing IgM seems to be predictive for the development of arthritis in patients with ECM. Serum immunoglobulin levels also fluctuate in relation to disease activity. Increased levels of serum IgM appear to correlate with increased joint and neurologic disease activity, while higher serum levels of IgG and IgA seem to correlate with remission.

In a series of 78 Lyme disease patients followed from the time of presentation throughout the course of their disease, all had circulating immune complexes (measured by I^{125} — CIq — binding assay) at the onset of their symptoms. It was found that circulating immune complex levels fluctuated along with disease activity, and that the rate at which they disappeared varied according to the organ systems involved. Specifically, in patients with ECM only, immune complex levels dropped rapidly as the rash resolved. Levels dropped more slowly in patients with joint involvement, and remained very high in those who eventually developed cardiac and CNS involvement — dropping very slowly as disease activity subsided. In patients with Lyme arthritis, immune complexes were always present in the synovial fluid at higher than serum levels.

Summary

Lyme disease appears to be an epidemic, systemic disease transmitted by certain species of the Ixodes tick complex. Skin, joints, myocardium and the central nervous system may be involved. Erythema chronicum migrans followed by the development of arthritis is the most common presentation of the disease. Immune complexes appear to mediate several, if not all, manifestations of the disease. The key to diagnosis is the recognition of the distinctive skin rash, ECM. An antecedent tick bite may be recalled, but a history of travel to an endemic area may suffice. The initial skin lesion responds to penicillin or tetracycline, and the incidence of subsequent arthritis is reduced by antibiotic administration. It is most likely confused with pauci-articular juvenile rheumatoid arthritis (JRA), but the uniform presence of distinctive rash, geographic and seasonal clustering, and occurrence in adults all make Lyme disease sufficiently distinguishable from JRA.

Self-Assessment Questions

For questions 1-4, one or more of the answers may be correct.

1. Which of the following historical items would be most helpful in making the diagnosis of Lyme disease?
 a. Recent travel to Long Island, New York
 b. Recent course of erythromycin for bronchitis
 c. Recent tick bite
 d. Stiff neck and photophobia for five days
2. Which of the following physical findings would be *most* helpful in making the diagnosis of Lyme disease?
 a. Splenomegaly
 b. Malar rash
 c. Annular rash with central clearing located on the right flank
 d. Bell's palsy
 e. Swollen, painful left knee
3. For a patient with Lyme disease, a ten-day course of oral tetracycline is most likely to ameliorate or prevent which of the following:
 a. Second degree A-V block
 b. Erythema chronicum migrans
 c. Aseptic meningitis
 d. Development of a swollen, tender knee joint
4. A 27-year-old white male with a recent history of ECM has recurrent syncopal episodes and is found to be in complete heart block. Appropriate therapy includes:
 a. PEN VK 500 mg orally 4 times daily for ten days
 b. Prednisone 60 mg orally per day for four days, tapering over 10-14 days
 c. ASA 975 mg orally 4 times per day for ten days
 d. Temporary pacemaker
5. The arthritis of Lyme disease is universally benign and self-limited. (T or F)
6. Serum levels of IgM are generally predictive of disease activity. (T or F)

(Answers on page 389)

Suggested Readings

1. Reik, L. *et al.:* Neurologic abnormalities of Lyme arthritis. *Medicine* 58:281-294, 1979.

2. Steere, A. C. *et al.:* Lyme arthritis. *Arthritis Rheum.* 20:7-17, 1977.

3. Steere, A.C. *et al.:* Erythema chronicum migrans and Lyme arthritis: The enlarging clinical spectrum. *Ann. Intern. Med.* 86:685-698, 1977.

4. Schrock, C. G.: Lyme disease: Additional evidence of widespread distribution. *Am. J. Med.* 72:700-702, 1982.



Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Drug Disposition in the Elderly

Question: Why do some elderly patients appear to be more ''sensitive'' to the therapeutic and adverse effects of drugs?

Answer: Drug reactions occur two to three times more often in older patients.[1, 2] This may reflect the practice of polypharmacy in the elderly and their increased susceptibility to drug interaction effects.

Physiologic changes due to aging and pathologic changes from disease can alter drug pharmacokinetics and modify drug effects. Some of these age related changes will be discussed briefly.

Absorption: Although most studies of drug absorption in the elderly are inconsistent, many suggest several age related changes that may alter drug absorption.[3-5] These include decreased splanchnic blood flow and motility and fewer absorbing cells. Gastric pH increases with age may appreciably alter solubility and thus the absorption of many drugs. Drugs absorbed by passive diffusion (aspirin, acetaminophen, sulfonamides) are absorbed normally through the gastrointestinal tract in the elderly.

Distribution: Changes in body composition associated with age may also influence drug distribution. With age there is a reduction in percentage of total body water, plasma volume, and extracellular fluid as the proportion of body fat increases.[7-8] This indicates that the aged patient may be smaller and has less muscle mass. Drugs distributed mainly in body water or lean body mass will result in increased blood levels if doses are not adjusted. Conversely, lipid soluble agents (sedatives) with an increased volume of distribution may accumulate and have prolonged duration of action.

Protein Binding: Several studies have shown that albumin levels decrease with age.[9-11] Although total protein levels remain the same, plasma albumin is about 20 per cent lower in the elderly patient. Less available albumin to bind drugs may result in increased free drug concentration.

Metabolism: The effects of aging on drug metabolism are difficult to predict due to wide individual variation and distribution changes. Age related decreases in liver enzyme activity have been documented,[12] but clinical significance has not been established.

Excretion: Changes in renal excretion of drugs (digoxin, procainamide) in the aged have been widely documented.[13-16] Renal function as measured by glomerular filtration rate (GFR) decreases by about 35 per cent between the ages of 20 and 80 years.[17] However, serum creatinine may remain ''normal'' due to a decreased muscle mass in the elderly.

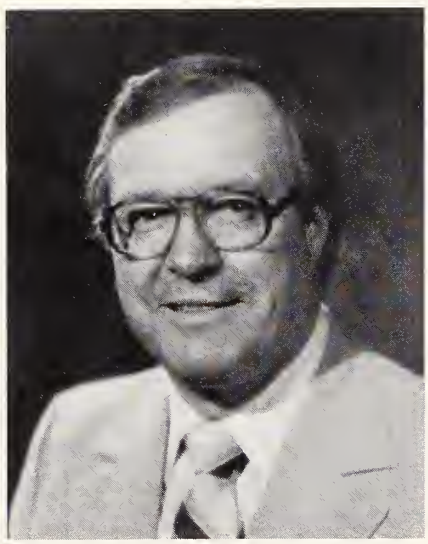
Receptor Sensitivity: The number of receptors per cell decreases with age.[18] Pharmacologic effects of drugs that act through clearly defined receptors may be altered in older individuals. This pertains to agents such as amphetamines, isoproterenol, atropine, and propranolol.

Dosage Selection: Most package inserts recommend dosage ranges for adults, children, and infants. Dosage selection for the elderly poses a problem due to a lack of comprehensive information in this area. In general, the elderly seem to require smaller dosages to achieve therapeutic effects.

As a group the elderly respond differently to drugs than do young people. Subtle physiological changes associated with aging may alter drug pharmacokinetics and modify drug effects. Consideration of the factors mentioned here can make prescribing for the elderly safer.

Submitted by
Joyce Generali, R.Ph.,
Pharmacy Resident

The President's Message



Dear Colleague:

This issue of *The Journal* is dedicated to geriatric medicine. A recent scientific session of our state society did an excellent job of discussing the aging process and the different therapeutic approaches necessary in caring for the elderly.

The increase in the geriatric population is in part a result of our improved medical knowledge and system of health care delivery. Biologically it is possible for many persons to reach the age of 95 years and as medical progress continues, the average life span approaches this apparent physiological limit. Better nutrition and environmental alterations have combined with improved medical care to create the impressive increase in this population that we now see.

From less than 21 million in 1972, the ranks of people over 65 years grew to 23 million in 1978, and will stand at 25 million by 1983. One-half of all people who have reached the age of 65 years are still living today, and by 1995 the elderly will number about 30 million. This number is expected to plateau at 38 million.

The main problem associated with the aged population does not consist of the increased numbers per se. Rather, it is such matters as the 80 per cent of health care dollars that are spent by 20 per cent of the people, most of these over 65 years or disabled. Among Medicare recipients, 40 per cent spend 90 per cent of Medicare dollars, of which 90 per cent is spent in the final two months of life.

These statistics show the important contribution of this age group to health care costs — a contribution that will only increase in the coming years. As physicians attempt to combat the large percentage of gross national product presently attributable to health care costs while maintaining standards of medical excellence, the over-65 population will continue to present a great challenge.

President



"Old Time Is Still A-flying"

It is well over 300 years since Robert Herrick issued his advice to young virgins regarding the gathering of rosebuds and unknowingly provided us with the warning of our title. Given the life expectancy of the 17th Century (which, we hasten to admit, we don't know), he might be surprised to find that today the fair flowers are not dying off as readily as they did. This refusal to pass away on the early schedule has resulted in a renaissance of interest in the matter of aging as it asserts itself firmly between its cause and effect, living and dying. The broad increase in life expectancy is a matter of record but the long view raises the question of whether aging has changed or is merely undergoing a chronologic deferment. In any event, the mercantile world is happily alert to the new market and there has been etymologic impact as well as the two words, geriatrics and gerontology, have come into everyday use.

Physicians are a little more comfortable, perhaps, with the former term since it relates specifically to their role in the medical management of aging. That role in many instances, however, is less sought than thrust upon them and the comfort stems more from a satisfactory adaptation to the medical requirements than a sense of accomplishment in dealing with the process. There is, if nothing else, a departure from the traditional etiology-diagnosis-therapy-cure pattern of other areas of medical service. Of more recent application, the term gerontology has provided a necessary extension of the concept of care for the aging since it encompasses not only the pathology but the psychobiology as well as the social and economic elements, and the nature of aging requires that the whole gamut of concerns be incorporated into the management. By this note, we are intentionally stressing the point that the cultural tenets regarding aging have an even longer history than the medical and have survived because of medicine's inherent inadequacies, not to mention economic necessity. Primitive societies, whose first interest was necessarily in survival, required of each member of the family community whatever service that individual could provide — from the small child gathering eggs or twigs for the fire to the old grandmother softening the hides by chewing on them until her teeth were gone. And there is something eminently practical in placing the elders out in the veldt or on the ice field to be taken by the predators or the elements when they were no longer able to perform some service for the group. Current standards of compassion lead us to hope the subjects were sufficiently imbued with the philosophy to find it an acceptable resolution of the situation. At least, we can read into it a certain dignity — more, in fact, than we have often accorded them in our supposedly enlightened societies.

Still our attitude toward aging and the aged (as well as others of undeveloped or lost capabilities) is frequently cited as an expression of our cultural humanitarianism. The current activities in the area are both quantitative — a response to that increased life expectancy with its consequent increase in the aging population — and qualitative — a part of the strong social winds of the times. It is these factors that impose a certain stress upon us as we contemplate the growing mass of elders since it requires a reexamination of some ingrained attitudes. We live in an age that celebrates youth and vitality, and aging is an affront to both. Delaying death or extending the aging process does not, unfortunately, mean an extension of youth despite the frantic efforts of some of the participants. As adjustments in standards have been made — employment policies, utilization of older persons in the social efforts and economy, for example — their implementation has been oriented to the fact that they must be made because the subjects are there to be dealt with regardless of capability, not that the capabilities have promoted the change. And in its own context, the medical profession, despite its front row seat, has had to struggle with its adjustment as much as has society generally.

Much is made these days of those particular con-

tributions the aging can make and of those individuals who are outstanding and challenging and inspiring examples of the elderly rendering valuable service. But reality requires that we remind ourselves, even as we seek ways for the aged to extend themselves, that such individuals are not the usual — they stand out and excite our admiration and attention because they *are* unusual. As specimens to try to emulate, they are marvels but we are still confronted with caring for an increasing multitude lacking in such capabilities or motivation.

Thus, the new concept may well put an unintended burden on the aging group. As a novice in that category, we can testify that there are aches and pains, both physical and mental, associated with the induction process. Not all can be attributed to lack of motivation to higher achievement or misbehaviors in earlier life or unambitious genes. Some of the more virtuous and abstemious or well-endowed members suffer greatly while the miscreants seem often to experience fewer — at least, no more — of these distresses. The earlier phases are usually greeted by acceptance of the lesser degrees and humorous references, a poorly disguised effort at denial. Nevertheless, the growing numbers of us testify to medical successes of the past, probably more, until recently at least, the public measures than private efforts. Moreover, the profession, partly to reduce medical costs and partly to accommodate a larger number of physicians than society can presumably absorb, has rediscovered preventive medicine and is taking steps to impose a state of general prophylaxis on the public. Whatever other benefits may come from this, experience teaches that its success will be measured in considerable degree by even greater growth of the elderly segment. Parenthetically, we can note that those attending the aged in the past were often of the same vintage as the patients — old comrades in the struggle, each reluctant to give the other up. But the patients are bidding to live longer than their physicians' effectiveness, and their care is falling to younger physicians. It will require a particular compassion on the part of the latter to compensate for this rapport produced by common age. They will be better equipped intellectually and technologically, but it remains to be seen, as indicated above, whether this means better health for the group or prolongation of the debilitation process.

To date, for example, it appears that greater progress has been made in the efforts toward physical support than mental. We have no intent to introduce a negative tone but wonder if the numerous studies inspired by the aging process have indicated how many bodies have been salvaged for survival (and cited as samples of success) while the minds continued their relentless deterioration. This brings into focus the ethical element of management — the same generically as that expressed in the "pulling the plug" controversies and contentions about the "quality of life." There is some irony in the fact that at a time when we are promoting the concept of patient involvement in the management of his case, we are also promoting an increasing population of persons who are incapable of such activity. Hard decisions will still devolve upon the family — or society — and physicians. Will the aged patient, begging for release rather than existence, be considered the proper agent in the decision — or subservient to social dictates?

Much of the uncertainty regarding the specifics of the future of aging stems from the lack of objective — particularly prospective — studies. Since we are the laboratory animals and our life cycles equal those of the investigators, it is impossible to compress into a short time meaningful alterations in handling which might lead to positive improvements. Furthermore, the current crop of elders, large though it is, is confronting conditions that are not those that applied to their grandparents or *their* antecedents, nor will they be the same for their progeny. This is a product of the progress being made in assessing the chemistry and biology and psychology of aging as well as the social usage of the aged, and brings increasing recognition that aging should not be viewed as an inexorable process for all to be shunted into uniformly and accepted unquestioningly on the basis of birthdays. We are daring to ask if the fact of existence must inevitably lead to a climax of distress as aging is accepted as a sentence from which diminished capabilities permit no appeal and death grants the only relief. Since medical purpose will of necessity be directed toward offering support and understanding as opposed to vigorous efforts to eradicate some disease, we must avoid the inclination to cloak those efforts in unrealistic pretense and unattainable expectations — either of our patients or ourselves. We must speak their language with directness or we will lose our audience — literally and figuratively.

We are accumulating a vast amount of basic information about the mergence of physiology and pathology as a function of time, about the immunologic and enzymatic complexities that provoke aging problems (or protect against them), and about the genetic dicta that regulate the underlying patterns. But the promise for the aged lies not just in the accumulation of medical information but the fact

(Continued on page 389)

First Class First Aid



Recommend

NEOSPORIN® Ointment

(POLYMYXIN B-BACITRACIN-NEOMYCIN)

- Broad-spectrum antibacterial
- Handy applicator tip



DESCRIPTION: Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and ½ oz and 1/32 oz (approx.) foil packets.

INDICATIONS: *Therapeutically* (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: • infected burns, skin grafts, surgical incisions, otitis externa • primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia) • secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis) • traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically,* the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: Not for use in the eyes or in the external ear canal if the eardrum is perforated. This product is contraindicated in those individuals who have shown hypersensitivity to any of its components.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

Wellcome / **Burroughs Wellcome Co.**
Research Triangle Park
North Carolina 27709

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(Continued from page 348)

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HYPERTENSION:



METHYLDOPA? RESERPINE? INDERAL? COUNTLESS THOUSANDS WOULD BE BETTER OFF WITH INDERAL® (PROPRANOLOL HCl) B.I.D.

The sooner, the better.

Today, INDERAL—instead of methyldopa, instead of reserpine.

INDERAL exhibits few of the disturbing side effects of methyldopa and reserpine. Sedation, depression, and impotence are rare.* Tolerance is not likely to occur, as it frequently does with methyldopa. For the vast majority of patients—INDERAL means a step toward improving the quality of life. (INDERAL should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.)*

INDERAL blocks beta-receptor sites *in the heart* to reduce heart rate and cardiac output—reducing cardiac work load—sparing an overburdened heart.

Hypertensive hearts can rest easy with INDERAL. For many—it is ideal, first-step therapy.

INDERAL—the sooner, the better for hypertension—a leading risk factor in coronary heart disease.[1]

*Please see following page for Brief Summary of Prescribing Information.



THE MOST WIDELY PRESCRIBED BETA BLOCKER IN THE WORLD

INDERAL®
(PROPRANOLOL HCl)
B.I.D. FOR HYPERTENSION

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
Inderal® BRAND OF **propranolol hydrochloride A beta-adrenergic blocking agent**

BEFORE USING INDERAL (PROPRANOLOL HYDROCHLORIDE), THE PHYSICIAN SHOULD BE THOROUGHLY FAMILIAR WITH THE BASIC CONCEPT OF ADRENERGIC RECEPTORS (ALPHA AND BETA), AND THE PHARMACOLOGY OF THIS DRUG

CONTRAINDICATIONS

INDERAL is contraindicated in 1) bronchial asthma; 2) allergic rhinitis during the pollen season; 3) sinus bradycardia and greater than first degree block; 4) cardiogenic shock; 5) right ventricular failure secondary to pulmonary hypertension; 6) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL; 7) in patients on adrenergic-augmenting psychotropic drugs (including MAO inhibitors), and during the two week withdrawal period from such drugs.

WARNINGS

CARDIAC FAILURE: Sympathetic stimulation is a vital component supporting circulatory function in congestive heart failure, and inhibition with beta-blockade always carries the potential hazard of further depressing myocardial contractility and precipitating cardiac failure. INDERAL acts selectively without abolishing the inotropic action of digitalis on the heart muscle (*i.e.*, that of supporting the strength of myocardial contractions). In patients already receiving digitalis, the positive inotropic action of digitalis may be reduced by INDERAL's negative inotropic effect. The effects of INDERAL and digitalis are additive in depressing AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, continued depression of the myocardium over a period of time can, in some cases, lead to cardiac failure. In rare instances, this has been observed during INDERAL therapy. Therefore, at the first sign or symptom of impending cardiac failure, patients should be fully digitalized and/or given a diuretic, and the response observed closely: a) if cardiac failure continues, despite adequate digitalization and diuretic therapy, INDERAL therapy should be immediately withdrawn; b) if tachyarrhythmia is being controlled, patients should be maintained on combined therapy and the patient closely followed until threat of cardiac failure is over.

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuation of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when INDERAL is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease, who are given propranolol for other indications.

IN PATIENTS WITH THYROTOXICOSIS, possible deleterious effects from long term use have not been adequately appraised. Special consideration should be given to propranolol's potential for aggravating congestive heart failure. Propranolol may mask the clinical signs of developing or continuing hyperthyroidism or complications and give a false impression of improvement. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. This is another reason for withdrawing propranolol slowly. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

IN PATIENTS DURING ANESTHESIA with agents that require catecholamine release for maintenance of adequate cardiac function, beta blockade will impair the desired inotropic effect. Therefore, INDERAL should be titrated carefully when administered for arrhythmias occurring during anesthesia.

IN PATIENTS UNDERGOING MAJOR SURGERY, beta blockade impairs the ability of the heart to respond to reflex stimuli. For this reason, with the exception of pheochromocytoma, INDERAL should be withdrawn 48 hours prior to surgery, at which time all chemical and physiologic effects are gone according to available evidence. However, in case of emergency surgery, since INDERAL is a competitive inhibitor of beta receptor agonists, its effects can be reversed by administration of such agents, *e.g.*, isoproterenol or levarterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in restarting and maintaining the heart beat has also been reported.

IN PATIENTS PRONE TO NONALLERGIC BRONCHOSPASM (*e.g.*, CHRONIC BRONCHITIS, EMPHYSEMA), INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETICS AND PATIENTS SUBJECT TO HYPOGLYCEMIA: Because of its beta-adrenergic blocking activity, INDERAL may prevent the appearance of premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia. This is especially important to keep in mind in patients with labile diabetes. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

USE IN PREGNANCY: The safe use of INDERAL in human pregnancy has not been established. Use of any drug in pregnancy or women of childbearing potential requires that the possible risk to mother and/or fetus be weighed against the expected therapeutic benefit. Embryotoxic effects have been seen in animal studies at doses about 10 times the maximum recommended human dose.

PRECAUTIONS

Patients receiving catecholamine depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine blocking action of this drug may then produce an excessive reduction of the resting sympathetic nervous activity. Occasionally, the pharmacologic activity of INDERAL may produce hypotension and/or marked bradycardia resulting in vertigo, syncopal attacks, or orthostatic hypotension.

As with any new drug given over prolonged periods, laboratory parameters should be observed at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function.

ADVERSE REACTIONS

Cardiovascular: bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, arterial insufficiency, usually of the Raynaud type, thrombocytopenic purpura.

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances, hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Miscellaneous: reversible alopecia. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been conclusively associated with propranolol.

Clinical Laboratory Test Findings: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DOSAGE AND ADMINISTRATION

ORAL

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 40 mg INDERAL twice daily, whether used alone or added to a diuretic. Dosage may be increased gradually until adequate blood pressure is achieved. The usual dosage is 160 to 480 mg per day. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

While twice-daily dosing is effective and can maintain a reduction in blood pressure throughout the day, some patients, especially when lower doses are used, may experience a modest rise in blood pressure toward the end of the 12 hour dosing interval. This can be evaluated by measuring blood pressure near the end of the dosing interval to determine whether satisfactory control is being maintained throughout the day. If control is not adequate, a larger dose, or 3 times daily therapy may achieve better control.

PEDIATRIC DOSAGE

At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

INTRAVENOUS

The intravenous administration of INDERAL has not been evaluated adequately in the management of hypertensive emergencies.

OVERDOSAGE OR EXAGGERATED RESPONSE

IN THE EVENT OF OVERDOSAGE OR EXAGGERATED RESPONSE, THE FOLLOWING MEASURES SHOULD BE EMPLOYED:

BRADYCARDIA—ADMINISTER ATROPINE (0.25 to 1.0 mg). IF THERE IS NO RESPONSE TO VAGAL BLOCKADE, ADMINISTER ISOPROTERENOL CAUTIOUSLY.

CARDIAC FAILURE—DIGITALIZATION AND DIURETICS.

HYPOTENSION—VASOPRESSORS, *e.g.*, LEVARTERENOL OR EPINEPHRINE (THERE IS EVIDENCE THAT EPINEPHRINE IS THE DRUG OF CHOICE).

BRONCHOSPASM—ADMINISTER ISOPROTERENOL AND AMINOPHYLLINE.

HOW SUPPLIED

INDERAL (propranolol hydrochloride)

TABLETS

No. 461—Each scored tablet contains 10 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

No. 462—Each scored tablet contains 20 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

No. 464—Each scored tablet contains 40 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

No. 468—Each scored tablet contains 80 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

INJECTABLE

No. 3265—Each ml contains 1 mg of propranolol hydrochloride in Water for Injection. The pH is adjusted with citric acid. Supplied as: 1 ml ampuls in boxes of 10.

Reference: 1. Freis, E. D. Hypertension (Suppl. II) *3*:230 (Nov.-Dec.) 1981

7997/482

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Lyme Disease

(Continued from page 379)

Answers

1. a, c
2. c
3. b, d
4. b, d
5. *False*
6. *True*

Register
to
VOTE

"Old Time Is Still A-flying"

(Continued from page 383)

that they are being considered a suitable inspiration for such efforts. The rejection of active care for the aged because they *are* aged is giving way to an increasing understanding that successful management lies not in guaranteeing a lasting "cure" — a patent impossibility — but in bringing the patient's capabilities, both physical and spiritual, to the most productive and comfortable level. And in each effort, there is a small step toward the next level of success.

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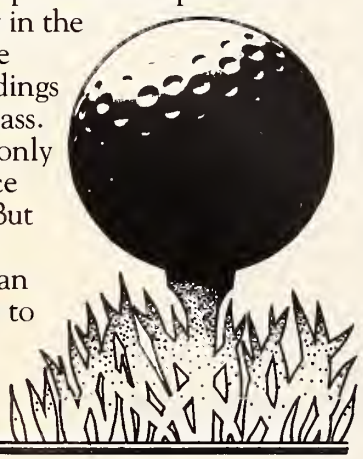
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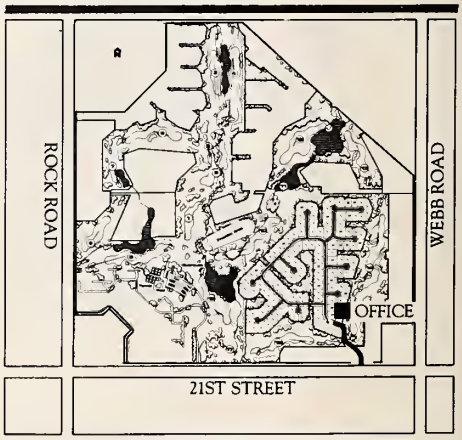
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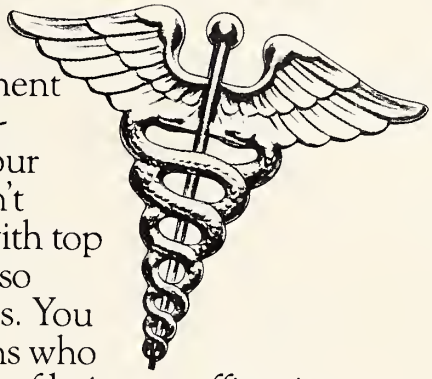


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(Continued from page 367)

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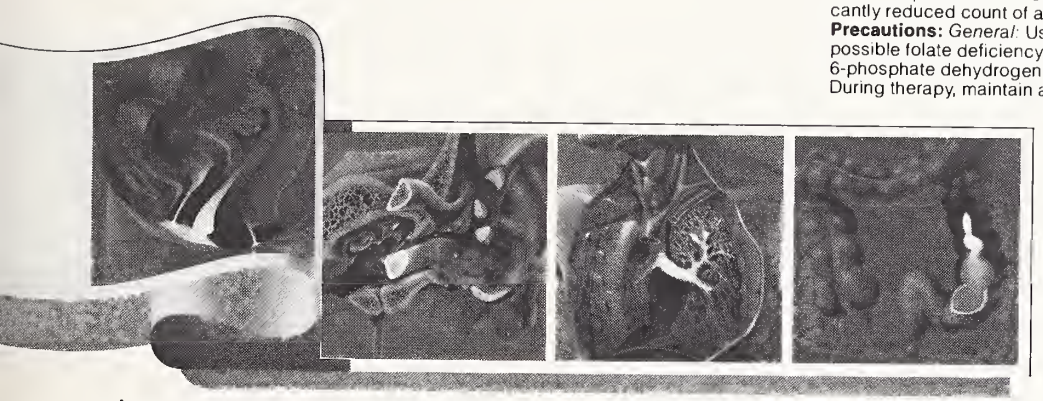
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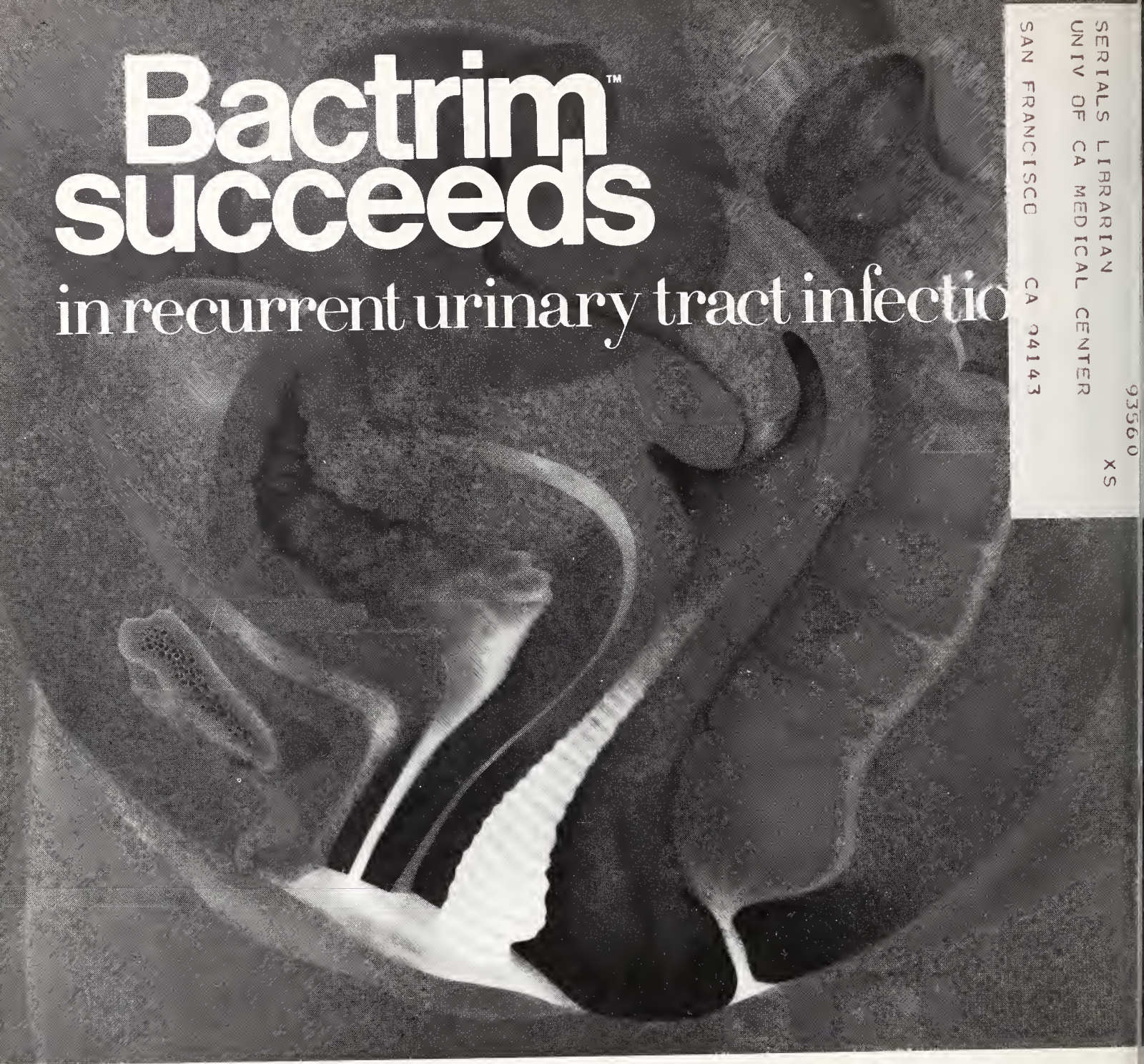
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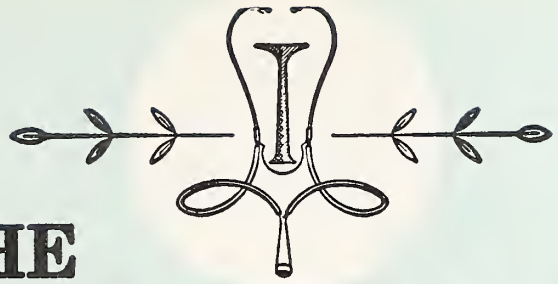
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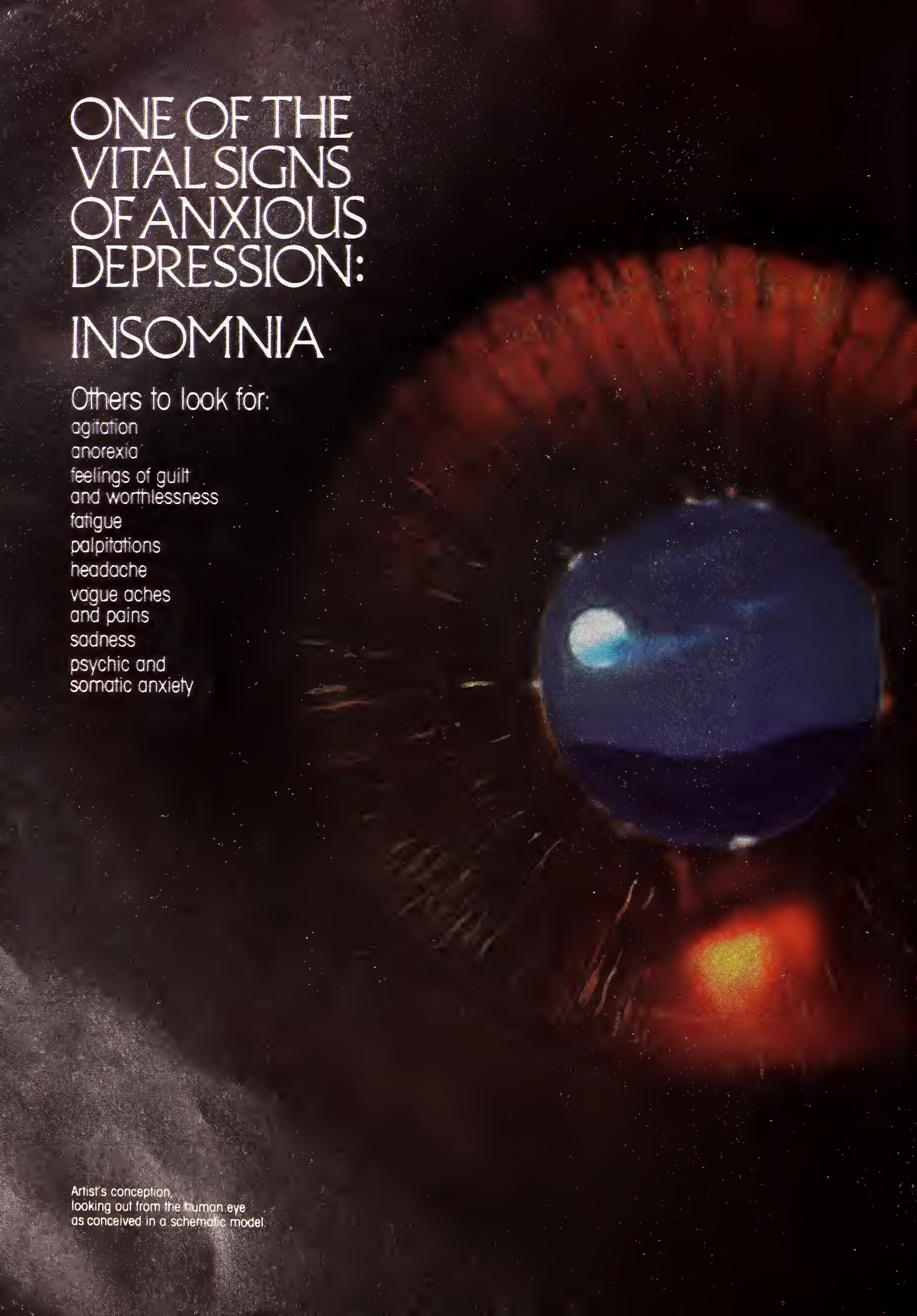
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Please see summary of complete product information on following page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).
Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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ep·i·dem·ic

/ep-a-'dem-ik/ i.e. Alcoholism 1. A disease affecting 10 million people in the United States. 2. Controlled through effective alcoholism treatment program.

Chances are, during a patient's preliminary examination, alcoholism is not one of your immediate suspicions. But with 10 million Americans suffering from this disease, and over 100,000 in the Kansas City area alone, perhaps it should be. Or perhaps the drinking problem is someone within your patient's family, causing them mental and even physical abuse.

At Bethany Medical Center's Alcoholism Rehabilitation Department (ARD), our professional staff offer both in- and outpatient programs for individuals suffering from alcoholism.

Our outpatient program is for those individuals whose affliction has not become debilitating, but recognize the need for treatment. It is specifically tailored to the individual's needs and incorporates family involvement.

Our inpatient program covers a 28-day phased treatment in a professional, yet unusual and cheery environment. Patients wear street clothes and participate in a busy, structured, daily routine that includes exercise, counseling, lectures and recreational time.

If you have a patient with a drinking problem, send for our detailed informational brochure, or personally contact one of our professional staff. Inpatient information: Sue Little, Director, Bethany Medical Center, 51 North 12th Street, K.C., KS. 66102. (913) 281-8951. Outpatient: Paul Anderson, Ph.D., Family Institute of Addiction Treatment, 7840 Washington Ave. Suite 300, K.C., KS. 66112. (913) 334-6300.

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Bethany Medical Center

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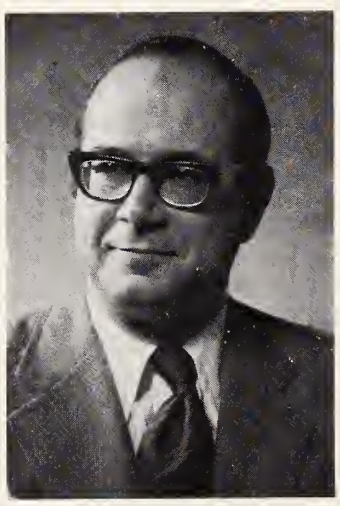
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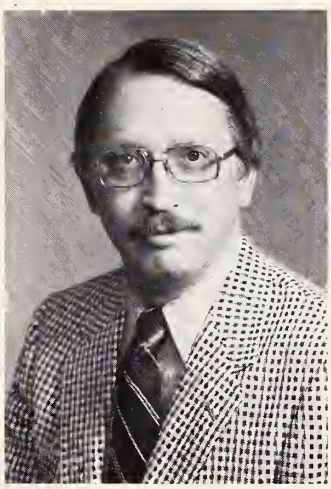
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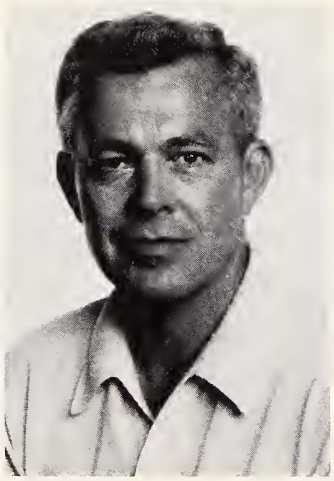
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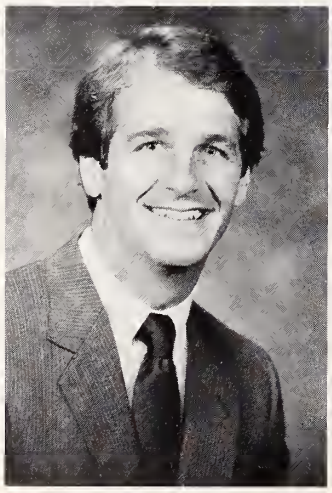


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LET'S TALK BUSINESS

A continuing series on business communication issues.

Increasing Productivity Through Information Management

The ability to increase productivity and remain competitive in the business office of the 80's will depend largely on how you manage information.

With the office consuming 40 to 50 percent of today's corporate expense dollar, nearly half the workforce deals with processing information rather than producing concrete products.

As a result, the tools of the 1800's – the typewriter, face-to-face meetings and even the telephone as we know it – are evolving into the systems which will make up the "office of the future."

Let's examine some of the ways that improvements in the management of information can begin to increase your productivity right away.

Increase Your Competitive Edge with Electronic Systems

To increase your competitive edge, you must improve on standard methods of operation. And electronic systems can help you. Too frequently, we simply accept traditional methods of communication.

Today's electronic communications systems have many time-saving features to help both large and small businesses. Automatic Callback, Call Forwarding, Call Waiting, Call Pickup and Automatic Route Selection are just a few of the features designed to minimize time wasted on callbacks and busy signals.

But the real advantage is system flexibility. Electronic telephone sets can be individually programmed so you can assign the right combination of features to each of your employees. And, you can make feature and station changes yourself – without the assistance of telephone company personnel. More long term, as technology advances or your needs change, software packages can be added to include new capabilities.

More Productive Meetings

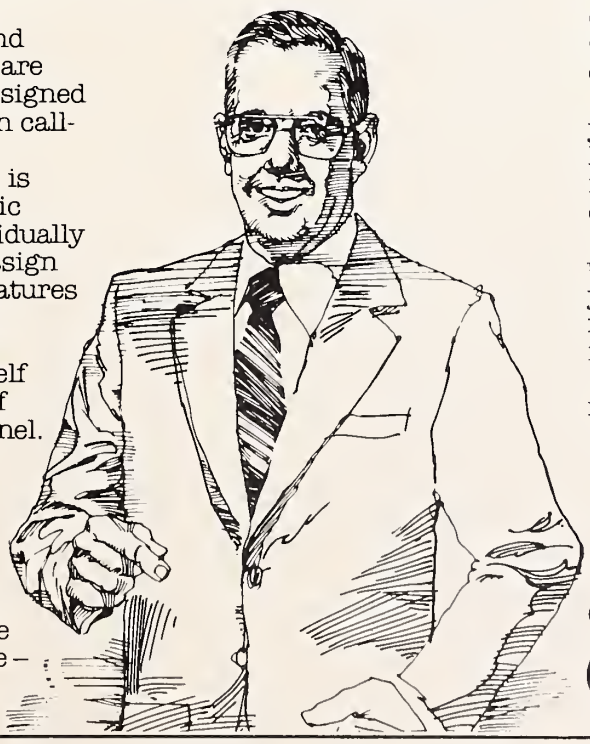
Meetings eat up a large amount of managerial time – especially when travel is involved.

While some face-to-face meetings will always be needed, in many cases, teleconferencing can help displace travel, reduce expenses and obtain immediately resolution to business problems.

Most multi-button and electronic systems have teleconferencing features for a minimum of three locations. And special telephones – as simple as an inexpensive speakerphone – can expand teleconferencing capabilities when several people are involved at a particular location.

A number of new teleconferencing products such as the high-quality GATT (Quorum* Group Audio Teleconferencing Terminal) can be installed in conference rooms for audio teleconferences involving large groups. And the Quorum Omnidirectional Microphone and Loudspeaker can enhance the transmission quality of GATT – or the speakerphone – even further.

Picturephone Meeting Service will become available in many cities in 1982. Besides enabling meeting participants to see and talk to each other, each video teleconference room will be equipped with cameras to display slides, charts and other graphic materials.



A New Approoch to Sales

Two components – technology and management systems, work together. And much of the technology that exists today is provided by Bell.

New telecommunications technology makes it possible to set up centers within a company to support and supplement the outside sales force. For example, a Telemarketing representative can respond to customers by using a data terminal to access preplanned questions that determine customer needs, find out shipping, billing and credit information and even make inventory adjustments.

WATS, 800 Service, automatic call distributors and data terminals are some of the ingredients needed to make a Telemarketing center work. Your Bell Account Executive can give you some help with management systems and procedures needed to actually get a Telemarketing program off the ground.

Stay Informed

You'll have questions from time to time regarding how to better manage your planned and existing communication systems. Obviously the entire subject could not be covered here.

So here's a suggestion: Call your Southwestern Bell Account Executive (or one of the toll-free numbers below) for more in-depth information.

You might even be able to use an accurate evaluation of your company's present and future communication needs – at no cost to you.

The way things are changing, it's worth the call.

Kenneth W. Fancher

Kenneth W. Fancher
Vice President, Business Sales

Call toll free: 1 800 643-8353.
(In Arkansas: 1 800 483-1223.)

Southwestern Bell

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An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor
Pulvules®, 250 and 500 mg

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coomb testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: Adverse effects considered related to cefaclor therapy are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70) and nausea and vomiting (1 in 90).

As with other broad-spectrum antibiotics, colitis, including rare instances of pseudomembranous colitis, has been reported in conjunction with therapy with Ceclor.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor® (cefaclor). Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [100281R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

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8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

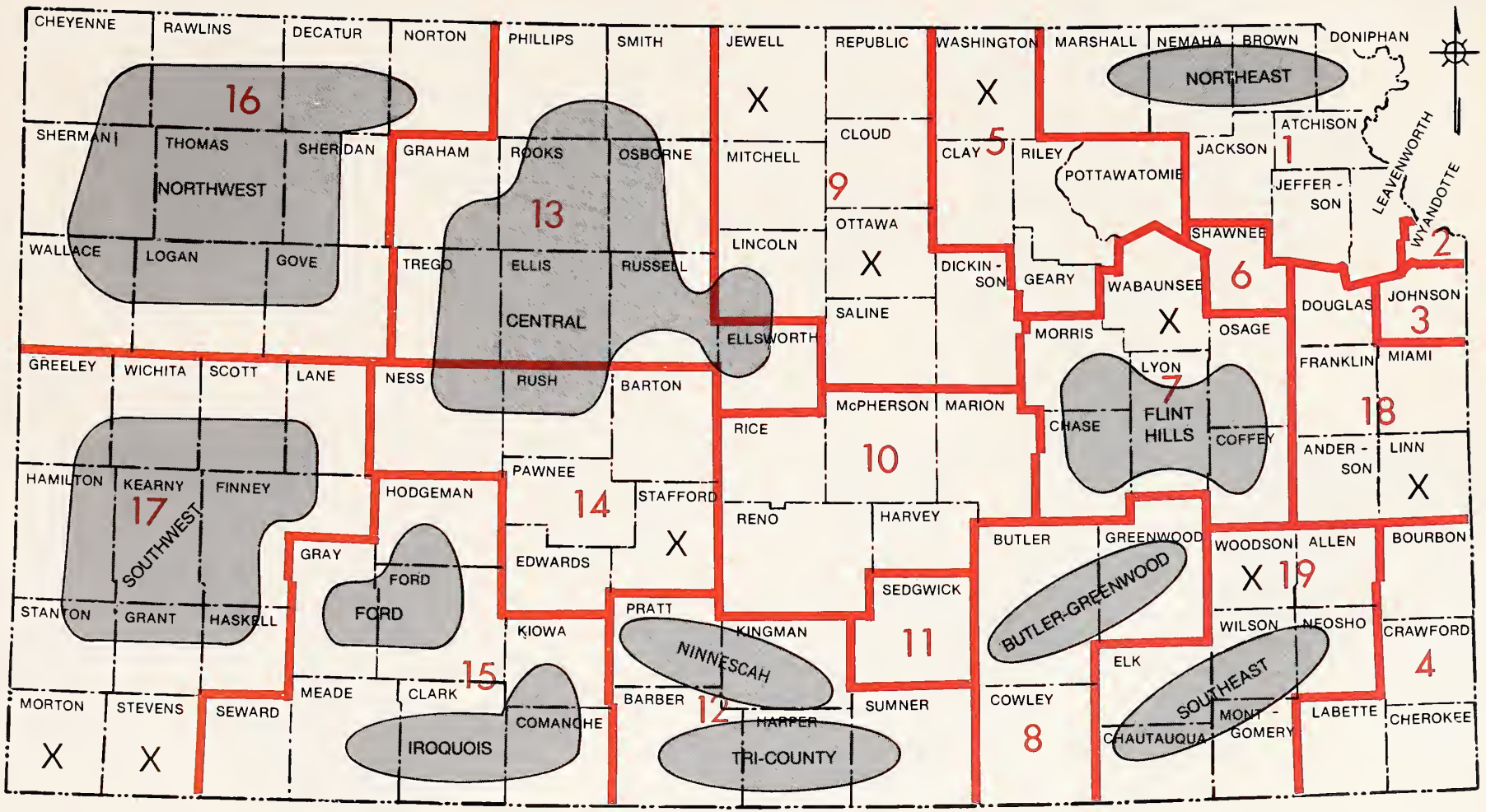
Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

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RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
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HYPERTENSION:



METHYLDOPA? RESERPINE? INDERAL? COUNTLESS THOUSANDS WOULD BE BETTER OFF WITH INDERAL® (PROPRANOLOL HCl) B.I.D.

The sooner, the better.

Today, INDERAL—instead of methyldopa, instead of reserpine.

INDERAL exhibits few of the disturbing side effects of methyldopa and reserpine. Sedation, depression, and impotence are rare.* Tolerance is not likely to occur, as it frequently does with methyldopa. For the vast majority of patients—INDERAL means a step toward improving the quality of life. (INDERAL should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.)*

INDERAL blocks beta-receptor sites *in the heart* to reduce heart rate and cardiac output—reducing cardiac work load—sparing an overburdened heart.

Hypertensive hearts can rest easy with INDERAL. For many—it is ideal, first-step therapy.

INDERAL—the sooner, the better for hypertension—a leading risk factor in coronary heart disease.[1]

*Please see following page for Brief Summary of Prescribing Information.



THE MOST WIDELY PRESCRIBED BETA BLOCKER IN THE WORLD

INDERAL®
(PROPRANOLOL HCl)
B.I.D. FOR HYPERTENSION

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
Inderal® BRAND OF **propranolol hydrochloride A beta-adrenergic blocking agent**

BEFORE USING INDERAL (PROPRANOLOL HYDROCHLORIDE), THE PHYSICIAN SHOULD BE THOROUGHLY FAMILIAR WITH THE BASIC CONCEPT OF ADRENERGIC RECEPTORS (ALPHA AND BETA), AND THE PHARMACOLOGY OF THIS DRUG.

CONTRAINDICATIONS

INDERAL is contraindicated in 1) bronchial asthma, 2) allergic rhinitis during the pollen season, 3) sinus bradycardia and greater than first degree block; 4) cardiogenic shock, 5) right ventricular failure secondary to pulmonary hypertension; 6) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL; 7) in patients on adrenergic-augmenting psychotropic drugs (including MAO inhibitors), and during the two week withdrawal period from such drugs.

WARNINGS

CARDIAC FAILURE: Sympathetic stimulation is a vital component supporting circulatory function in congestive heart failure, and inhibition with beta-blockade always carries the potential hazard of further depressing myocardial contractility and precipitating cardiac failure. INDERAL acts selectively without abolishing the inotropic action of digitalis on the heart muscle (*i.e.*, that of supporting the strength of myocardial contractions). In patients already receiving digitalis, the positive inotropic action of digitalis may be reduced by INDERAL's negative inotropic effect. The effects of INDERAL and digitalis are additive in depressing AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, continued depression of the myocardium over a period of time can, in some cases, lead to cardiac failure. In rare instances, this has been observed during INDERAL therapy. Therefore, at the first sign or symptom of impending cardiac failure, patients should be fully digitalized and/or given a diuretic, and the response observed closely: a) if cardiac failure continues, despite adequate digitalization and diuretic therapy, INDERAL therapy should be immediately withdrawn; b) if tachyarrhythmia is being controlled, patients should be maintained on combined therapy and the patient closely followed until threat of cardiac failure is over.

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuation of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when INDERAL is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease, who are given propranolol for other indications.

IN PATIENTS WITH THYROTOXICOSIS, possible deleterious effects from long term use have not been adequately appraised. Special consideration should be given to propranolol's potential for aggravating congestive heart failure. Propranolol may mask the clinical signs of developing or continuing hyperthyroidism or complications and give a false impression of improvement. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. This is another reason for withdrawing propranolol slowly. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

IN PATIENTS DURING ANESTHESIA with agents that require catecholamine release for maintenance of adequate cardiac function, beta blockade will impair the desired inotropic effect. Therefore, INDERAL should be titrated carefully when administered for arrhythmias occurring during anesthesia.

IN PATIENTS UNDERGOING MAJOR SURGERY, beta blockade impairs the ability of the heart to respond to reflex stimuli. For this reason, with the exception of pheochromocytoma, INDERAL should be withdrawn 48 hours prior to surgery, at which time all chemical and physiologic effects are gone according to available evidence. However, in case of emergency surgery, since INDERAL is a competitive inhibitor of beta receptor agonists, its effects can be reversed by administration of such agents, *e.g.*, isoproterenol or levarterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in restarting and maintaining the heart beat has also been reported.

IN PATIENTS PRONE TO NONALLERGIC BRONCHOSPASM (*e.g.*, CHRONIC BRONCHITIS, EMPHYSEMA), INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

DIABETICS AND PATIENTS SUBJECT TO HYPOGLYCEMIA: Because of its beta-adrenergic blocking activity, INDERAL may prevent the appearance of premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia. This is especially important to keep in mind in patients with labile diabetes. Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure.

USE IN PREGNANCY: The safe use of INDERAL in human pregnancy has not been established. Use of any drug in pregnancy or women of childbearing potential requires that the possible risk to mother and/or fetus be weighed against the expected therapeutic benefit. Embryotoxic effects have been seen in animal studies at doses about 10 times the maximum recommended human dose.

PRECAUTIONS

Patients receiving catecholamine depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine blocking action of this drug may then produce an excessive reduction of the resting sympathetic nervous activity. Occasionally, the pharmacologic activity of INDERAL may produce hypotension and/or marked bradycardia resulting in vertigo, syncopal attacks, or orthostatic hypotension.

As with any new drug given over prolonged periods, laboratory parameters should be observed at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function.

ADVERSE REACTIONS

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; arterial insufficiency, usually of the Raynaud type; thrombocytopenic purpura.

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Miscellaneous: reversible alopecia. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been conclusively associated with propranolol.

Clinical Laboratory Test Findings: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DOSAGE AND ADMINISTRATION

ORAL

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 40 mg INDERAL twice daily, whether used alone or added to a diuretic. Dosage may be increased gradually until adequate blood pressure is achieved. The usual dosage is 160 to 480 mg per day. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

While twice-daily dosing is effective and can maintain a reduction in blood pressure throughout the day, some patients, especially when lower doses are used, may experience a modest rise in blood pressure toward the end of the 12 hour dosing interval. This can be evaluated by measuring blood pressure near the end of the dosing interval to determine whether satisfactory control is being maintained throughout the day. If control is not adequate, a larger dose, or 3 times daily therapy may achieve better control.

PEDIATRIC DOSAGE

At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

INTRAVENOUS

The intravenous administration of INDERAL has not been evaluated adequately in the management of hypertensive emergencies.

OVERDOSAGE OR EXAGGERATED RESPONSE

IN THE EVENT OF OVERDOSAGE OR EXAGGERATED RESPONSE, THE FOLLOWING MEASURES SHOULD BE EMPLOYED:

BRADYCARDIA—ADMINISTER ATROPINE (0.25 to 1.0 mg). IF THERE IS NO RESPONSE TO VAGAL BLOCKADE, ADMINISTER ISOPROTERENOL CAUTIOUSLY.

CARDIAC FAILURE—DIGITALIZATION AND DIURETICS.

HYPOTENSION—VASOPRESSORS, *e.g.*, LEVARTERENOL OR EPINEPHRINE (THERE IS EVIDENCE THAT EPINEPHRINE IS THE DRUG OF CHOICE).

BRONCHOSPASM—ADMINISTER ISOPROTERENOL AND AMINOPHYLLINE.

HOW SUPPLIED

TABLETS — INDERAL (propranolol hydrochloride)

No. 461—Each scored tablet contains 10 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

No. 462—Each scored tablet contains 20 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

No. 464—Each scored tablet contains 40 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

No. 468—Each scored tablet contains 80 mg of propranolol hydrochloride, in bottles of 100 and 1,000. Also in unit dose package of 100.

INJECTABLE

No. 3265—Each ml contains 1 mg of propranolol hydrochloride in Water for Injection. The pH is adjusted with citric acid. Supplied as: 1 ml ampuls in boxes of 10.

Reference: 1. Freis, E. D.: Hypertension (Suppl. II) 3:230 (Nov.-Dec.) 1981

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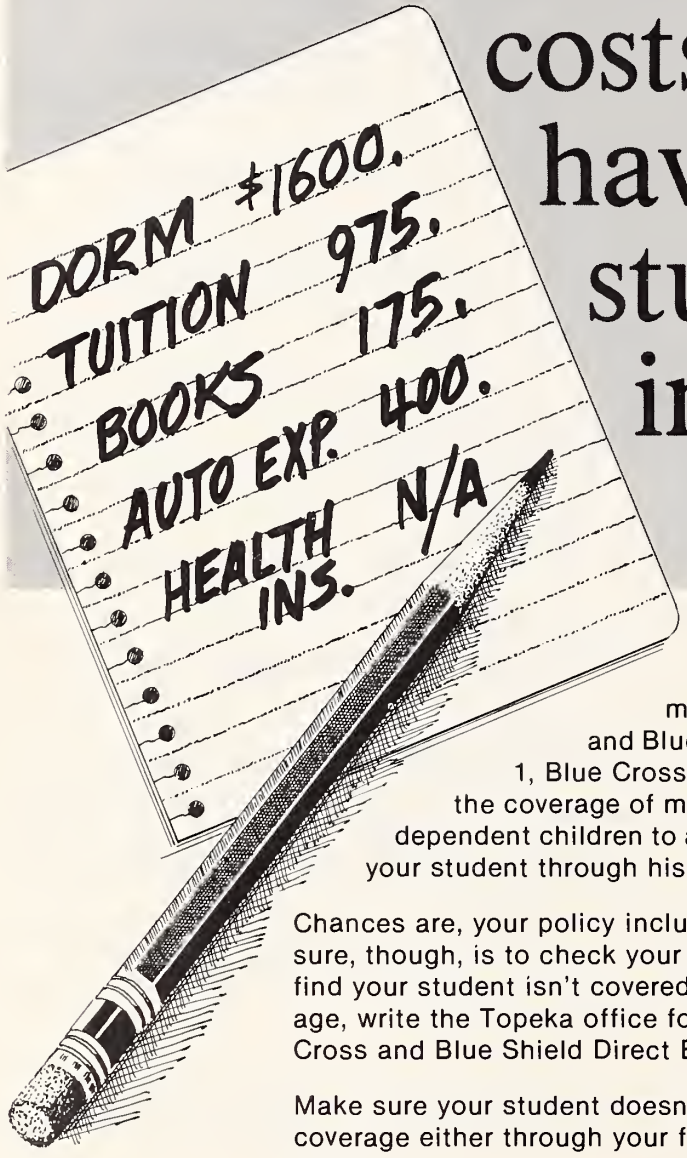
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Chances are, your policy includes this new benefit. The only way to be sure, though, is to check your Blue Cross and Blue Shield policy. If you find your student isn't covered under your plan because of marriage or age, write the Topeka office for information regarding the special Blue Cross and Blue Shield Direct Enrolled Student Program.

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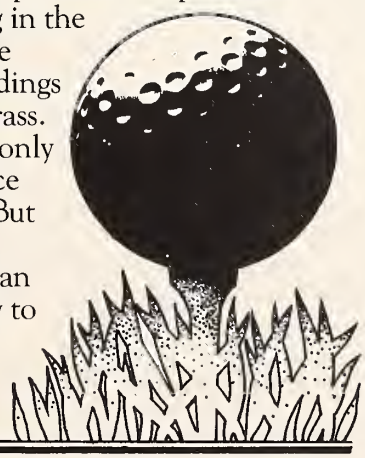
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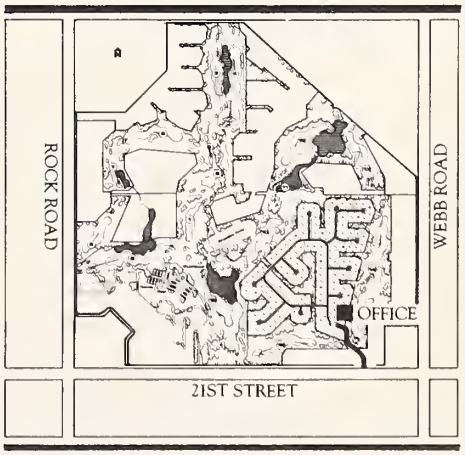
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ON-LINE PATIENT INQUIRY saves clerical time, improves patient relations and increases practice efficiency by providing immediate answers on patient accounts. Eliminates the need for cross-reference files.

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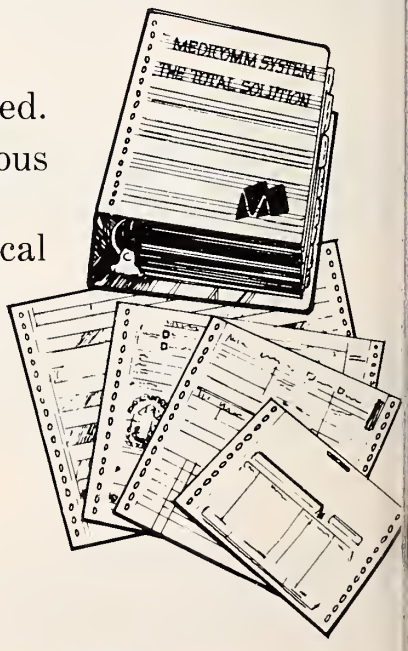
AUTOMATED STATEMENT GENERATION reduces tedious manhours and increases cash flow.

COMPREHENSIVE INSURANCE BILLING reduces clerical time and increases cash flow.

TAILORED DUNNING MESSAGES AND COLLECTION LETTERS increase the collection of past due accounts and reduces write-offs.

FAMILY BILLING saves clerical cost in statement preparation; posting of payments and postage.

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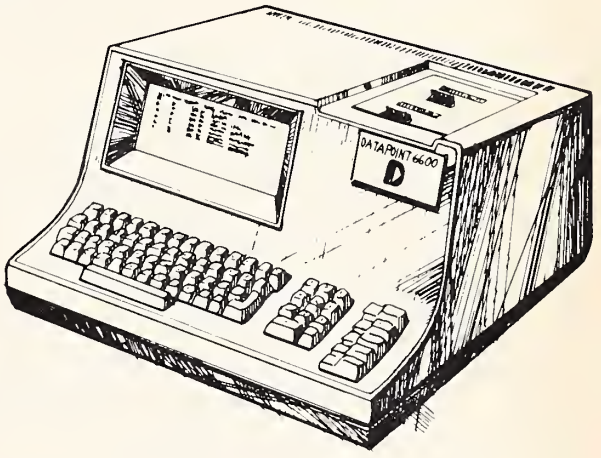


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ON-LINE IN-HOUSE SYSTEM eliminates the need for many bulky and costly print-outs and eliminates the need to send your records outside.

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- Open-item and balance-forward statements during the same cycle
- Statements by account number or zip code
- Print options
 - — Tailored dunning or greeting message
 - — Comment for each charge posted (25 char, one time only)
 - — Patient discounts, contract payments due, or service charges generated
 - — Layman's description of service rather than medical terminology which prints on the insurance forms
 - — Controls on printing credit balance, zero balance, or minimum balance statements
- Family billing allows up to 36 family members under one account. Posting and inquiry by Medical Record Number

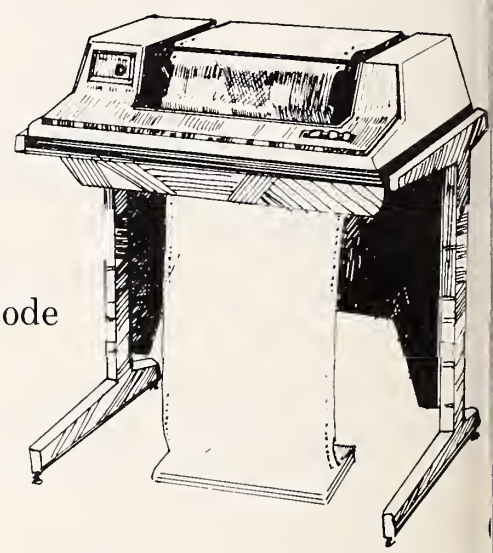
Insurance Processing

- Medicare, Medicaid, Blue Cross/Blue Shield, AMA and other claims forms produced on request or by cycle code
- On-line insurance printing on AMA forms
- Cross-references up to five procedure coding schemes
- Secondary insurance claims filed simultaneously with primary or suspended, pending payment from primary
- Automatic generation of claim numbers
- Disallowed charges on assigned claims automatically written off
- Updating of last insurance filed date for each account
- "Insurance claim filed" messages written to the accounts receivable file for each account
- Other variations in insurance printing such as signature on file, assignment of benefits, varying provider ID's for different insurance carriers, diagnosis codes, places of treatment, places of service, procedure modifier codes, referring physicians, hospital admission and discharge dates, and many others

Transaction Posting

- On-line or overnight posting of charges, receipts and adjustments
- On-line batch balancing and error correction
- Visual verification of all data — descriptions, names, prices are displayed to aid the user

Reporting



- Revenue and Service Analysis — Physicians Productivity
- Aged Accounts Receivable Analysis (to over 120 days) format options:

 by account — no detail
 by account — current transactions
 by account — complete history
 and summary totals by aged category/account type/billing code
- Insurance Claim Status
- Production Summary by Type of Service
- Hospital Census
- Recall Notices
- Demographic or Transaction Report Generator
 - — Selection on most data variables including account type, bill code, bill cycle, balance, insurance carriers, dates of last payment, financial class, procedure codes, diagnosis codes, physician
 - — User defined sort sequence (primary, secondary, tertiary), User defined breaks (page, double space)

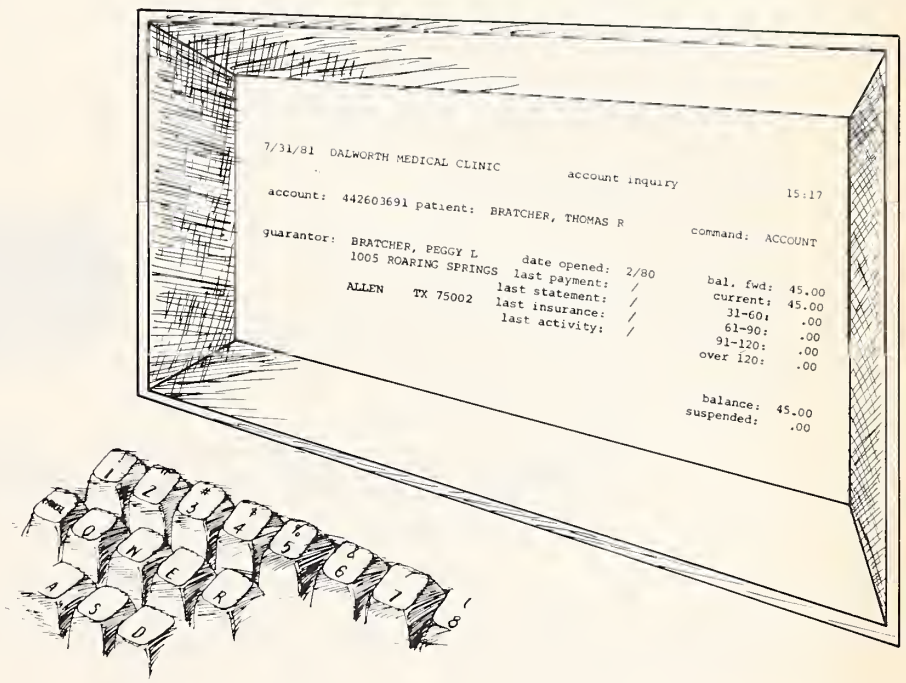
On-line Inquiry, Real-time Editing & Maintenance, Listing

- Patient master file — demographic, insurance and financial
- Practice and processing control — user defined — account types, bill codes, bill cycles, financial classes, billing control codes, departments and physicians, places of treatment, pricing by doctor or department, messages, collection letters
- Procedure and diagnosis codes and descriptions
- Account numbers — user defined or optional automatic generation and check digits
- Employers, insurance carriers, insurance claims
- Alphabetically by guarantor or patient
- History and current transactions
- Two insurance carriers with group and certificate identification and name of insured
- Comment — 10 lines/25 characters each for miscellaneous data
- Contract payment definition and terms

- Promises-to-pay — payment amount and due date
- Patient recall — automatic calculations of next due date
- Hospital admission, discharge, and room number

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- Worker's compensation
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- Accounting packages
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 - — Accounts Payable
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Arkansas City — Arkansas City Memorial, 216 West Birch 67005, Stephen Mason, Adm. — 316/442-2500

Ashland — Ashland District, 709 Oak 67831, Hazel Thomas, Adm. — 316/635-2241

Atchison — Atchison, 1301 N. Second 66002, James A. Asher, Adm. — 913/367-2131

Attica — Attica District, P.O. Box 268 67009, Ron McCoy, Adm. — 316/254-7253

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Baxter Springs — Baxter Memorial, P.O. Box 151 66713, James L. Kirkpatrick, Adm. — 316/856-2314

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Clay Center — Clay County, 617 Liberty 67432, Jon J. Robinson, Adm. — 913/632-2144

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Fredonia — St. Margaret's Mercy, P.O. Box 519 66736, Sister Mary Faith Matney, Adm. — 316/378-2121

Garden City — St. Catherine, 608 N. Fifth Street 67846, Sister Anita Schugart, Pres. — 316/275-6111

Gardner — Gardner Community Medical Center, 427 W. Main 66030, Henry R. Steinhaus, Adm. — 913/884-8711

Garnett — Anderson County, P.O. Box 309 66032, Randall Hempling, Adm. — 913/448-3131

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Harper — Harper County District #5, 12th & Maple 67058, Marshall P. Ray, Adm. — 316/896-7324

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Herington — Herington Municipal, 100 East Helen 67449, Curt Thomas, Adm. — 913/258-2207

Hiawatha — Hiawatha Community, 300 Utah 66434, Gerald E. Alkire, Adm. — 913/742-2131

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Hoisington — Hoisington Lutheran, 250 West Ninth 67544, Angeline C. Deutsch, Adm. — 316/653-2114

Holton — Holton City, 418 West Fifth 66436, Roger S. Lewis, Adm. — 913/364-2116

Horton — Horton Community, P.O. Box 191 66439, David Bauman, Adm. — 913/486-2642

Hoxie — Sheridan County, 826 Eighteenth Street 67740, Elizabeth Charlton, R.N., Adm. — 913/675-3281

Hugoton — Stevens County, P.O. Box 10 67951, James Mackay, Jr., Adm. — 316/544-8511

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Kansas City — University of Kansas Medical Center, 39th & Rainbow Blvd. 66103, Dr. Masahiro Chiga, Med. Dir. — 913/588-5000

Kingman — Kingman Community, P.O. Box 376 67068, Phillip L. Unruh, Adm. — 316/532-3147

Kinsley — Edwards County, P.O. Box 99 67547, Robert L. Mullen, Adm. — 316/659-3621

Kiowa — Kiowa District, 810 Drumm Street 67070, Buck McKinney, Adm. — 316/825-4130

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Lakin — Kearny County, 306 Kansas 67860, Jerrell J. Horton, Adm. — 316/355-7111

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Lawrence — Lawrence Memorial, 325 Maine Street 66044, Robert B. Ohlen, Chief Executive Officer — 913/843-3680

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Leavenworth — Cushing Memorial, 623 Marshall 66048, Charles L. Rogers, Adm. — 913/682-8000

Leavenworth — Saint John, 3500 South 4th 66048, Sister Mary Concetta Mock, Acting Adm. — 913/682-3721

Leoti — Wichita County, P.O. Box 968 67861, Kim Berning, Adm. — 316/375-2233

Liberal — Southwest Medical Center, P.O. Box 1340 67901, William Griffith, Adm. — 316/624-1651

Lincoln — Lincoln County, 624 North Second 67455, Larry Baecht, Adm. — 913/524-4403

Lindsborg — Lindsborg Community, 605 West Lincoln 67456, Marie Muller, R.N., Adm. — 913/227-3308

Lyons — Rice County District #1, 619 South Clark 67554, Tom Talley, Adm. — 316/257-2348

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Manhattan — Memorial, P.O. Box 1208 66502, Thomas O. Faulkner, Exec. Dir. — 913/776-3300

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Newton — Bethel Deaconess, 411 Southeast Second 67114, Marvin H. Ewert, Adm. — 316/283-2700

Newton — Prairie View Mental Health Center, P.O. Box 467 67114, Larry W. Nikkel, Adm. — 316/283-2400

Norton — Norton County, P.O. Box 250 67654, Richard Miller, Adm. — 913/877-3351

Norton — Valley Hope Alcoholism Treatment Center, P.O. Box 410 67654, Dennis R. Gilhousen, Adm. — 913/877-5111

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Olathe — Mid-Continent Psychiatric, 122 North Cooper 66061, Jack Rash, Adm. — 913/782-4282

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Onaga — Community, 6th & Lucien 66521, James M. Cazier, Adm. — 913/889-4274

Osborne — Osborne County Memorial, 424 West New Hampshire 67473, Dona Stephenson, R.N., Adm. — 913/346-2121

Oswego — Oswego, Rt. 2, Box 10A 67356, Patrick Lawrence, Adm. — 316/795-2921

Ottawa — Ransom Memorial, 13th & South Main 66067, Gary A. Moore, Adm. — 913/242-3344

Overland Park — Suburban Medical Center, P.O. Box 5959 66215, Jack Salberg, Exec. Dir. — 913/492-1000

Paola — Miami County, 501 South Hospital Drive 66071, Robert E. Johnson, Adm. — 913/294-2327

Parsons — Labette County Medical Center, P.O. Box 767 67357, Jerry D. Lilley, Adm. — 316/421-4880

Parsons — Katy Memorial, 400 Katy 67357, Leslie Hammett, Assoc. Adm. — 316/421-2700

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St. John — St. John District, 609 East 1st 67576, Art Thomas, Adm. — 316/549-3255

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WaKeeney — Trego County-Lemke Memorial, 320 Thirteenth Street 67672, Jim Wahlmeir, Adm. — 913/743-2134
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Wellington — Wellington Hospital & Clinic, 924 South Washington Avenue 67152, Regina E. Cantrell, Adm. — 316/326-3353
Westmoreland — Dechairo Hospital, Inc., First & North Streets 66549, Donn Demaree, Adm. — 913/457-3311
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Wichita — St. Joseph Medical Center, 3600 East Harry Street 67218, Joseph A. Heeb, Adm. — 316/685-1111
Wichita — Wesley Medical Center, 550 North Hillside 67214, Roy C. House, Pres. and Chief Executive Officer — 316/688-2468
A. B. Davis, Jr., Exec. V.P. and Chief Operating Officer — Ext. 7114
Winchester — Jefferson County Memorial, 66097, Ben Witzke, Adm. — 913/774-4340
Winfield — William Newton Memorial, 1300 East 5th 67156, Harold W. Steadham, Adm. — 316/221-2300

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Larned — Larned State Hospital, P.O. Box 89 67550, Mrs. Hildreth Hultine, Supt. — 316/285-2131
Norton — Norton State Hospital, 67654, Sam H. Freeland, Supt. — 913/877-3301
Osawatomie — Osawatomie State Hospital, P.O. Box 500 66064, J. Russell Mills, Supt. — 913/755-3151
Parsons — Parsons State Hospital & Training Center, P.O. Box 738 67357, Gary Daniels, M.D., Supt. — 316/421-6550
Topeka — Kansas Neurological Inst., 3107 West 21st Street 66604, Ann M. Marshall, Ph.D., Supt. — 913/296-5301
Topeka — Topeka State Hospital, 2700 West 6th 66606, Eberhard G. Burdzik, M.D., Supt. — 913/296-4307
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Fort Riley — Irwin Army Comm. Hospital, 66442, Major Jerry Thompson, Chief, Patient Adm. Division — 913/239-3663
Leavenworth — Veterans Administration Center, 66048, Margaret C. Michelson, Center Director — 913/682-2000

Topeka — Veterans Administration Medical Center, 2200 Gage 66622, Paul K. Kennedy, Medical Center Director — 913/272-3111
Wichita — McConnell Air Force Base, USAF Hospital 67221, Major Joseph A. Borho, Hosp. Adm. — 316/685-9254
Wichita — Veterans Administration Center, 5500 E. Kellogg 67218, George B. Lappin, Dir. — 316/685-2221

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Dodge City — Trinity Hospital — 316/483-8133
Emporia — Newman Memorial Hospital — 316/342-7120
Fort Scott — Mercy Hospital — Day: 316/223-3100; Night: 316/223-2200
Great Bend — Central Kansas Medical Center — Day: 316/793-3523; Night: 316/792-2511
Hays — Hadley Memorial Hospital — Day: 913/625-2515, Ext. 237; Night: 913/625-3441
Kansas City
University of Kansas Medical Center — 913/588-6633
Bethany Hospital — 913/621-6600
Lawrence — Lawrence Memorial Hospital — Day: 913/843-3680 or 842-4477; Night: 913/843-5874
Parsons — Labette County Medical Center — 316/421-4880
Salina — St. John's Hospital — 913/827-5591, Ext. 222
Topeka — Stormont-Vail Hospital — 913/354-6100
Wichita — Wesley Hospital — 316/688-2222
Kansas Poison Control Information Center:
Kansas Department of Health, Food and Drug Division, Topeka — Evan Wright, Director — 913/862-9360, Ext. 541
Antivenin Index Center — 405/271-5454

HOME HEALTH AGENCIES

Anderson County Hospital, Garnett 60032 — 913/448-3131
Barton County Health Dept., Courthouse, Great Bend 67530 — 316/793-7879
Butler-Greenwood County Health Dept., Courthouse, El Dorado 67042 — 316/321-3400
Clay County Hospital, Clay Center 67432 — 913/632-2144
Cloud County Health Dept., Courthouse, Concordia 66901 — 913/243-3588
Coffey County Health Dept., Courthouse, Burlington 66839 — 316/364-5831
Douglas County Visiting Nurses' Association, 342 Missouri St., Lawrence 66044 — 913/843-3738
Ellis County-St. Anthony Hospital, Hays 67601 — 913/625-2556
Ellsworth County Health Dept., Courthouse, Ellsworth 67439 — 913/472-4234
Ford County Health Dept., Courthouse, Dodge City 67801 — 316/225-4991
Franklin County Health Dept., 112 W. Tecumseh, Ottawa 60667 — 913/242-1873
Graham County Hospital, Hill City 67642 — 913/674-2121
Harper County Health Dept., Courthouse, Anthony 67003 — 316/842-5264
Harvey County Health Dept., Courthouse, Newton 67114 — 316/283-1060
Jackson County Health Dept., Courthouse, Holton 66436 — 913/364-2670
Jefferson County Health Dept., Courthouse, Oskaloosa 66066 — 913/863-2447
Kansas City Visiting Nurses' Association, One Gateway Center, Suite 219, Kansas City, Kansas 66117 — 913/371-3770
Kingman County Health Dept., Courthouse, Kingman, 67068 — 316/532-2221
Labette County Health Dept., Box 786, Parsons 67357 — 316/421-4350
Logan County Hospital, Oakley 67748 — 913/672-3211
Lyon County Home Health Service, Newman Memorial Hospital, Emporia 66801 — 316/342-8562
McPherson County Health Dept., Courthouse, McPherson 67460 — 316/241-1753
Morris County Health Dept., Courthouse, Council Grove 66846 — 316/767-5175
Osage County Health Dept., Courthouse, Lyndon 66451 — 913/828-3281
Pratt County Health Dept., 106 E. 2nd, Pratt 67124 — 316/672-6122
Reno County-St. Elizabeth Hospital, 500 W. 20th, Hutchinson 67501 — 316/665-5531
Republic County Health Dept., Courthouse, Belleville 66935 — 913/527-5385
Rice County Health Dept., Courthouse, Lyons 67554 — 316/257-2171
Riley County Health Dept., 616 Poyntz, Manhattan 66502 — 913/776-9721
Russell County Health Dept., Courthouse, Russell 67665 — 913/483-2641
Salina-Saline County Health Dept., 300 W. Ash, Salina 67401 — 913/827-9376

Sedgwick County Health Dept., 1900 E. 9th, Wichita 67214 — 316/268-;8433

Shawnee County Health Dept., 1615 W. 8th, Topeka 66606 — 913/233-8961

Sheridan County Hospital, Hoxie 67740 — 913/675-3281

Thomas County-St. Thomas Hospital, Colby 67701 — 913/462-3335

Washington County Health Dept., Washington 66968 — 913/325-2600

GENETIC COUNSELING CENTERS

Kansas City — Genetic Counseling Center, Division of Medical Genetics, K.U.M.C., 39th & Rainbow Blvd., Kansas City, KS 66103 — 913/588-6043 — R. Neil Schimke, M.D., Director

Topeka — Genetic Counseling Center, 2029 McAlister, Topeka, KS 66604 — 913/273-6173 — David E. Gray, M.D., Director

Wichita — Genetic Counseling Unit, Wesley Medical Center, 550 N. Hillside, Wichita, KS 67214 — 316/688-2468 — Sechin Cho, M.D., Director

To Contact Your Legislators:

U. S. CONGRESSIONAL DELEGATION

Senators:

Robert Dole, 2213 Dirksen Senate Office Bldg., 20510. (202) 224-6521

Nancy L. Kassebaum, 304 Russell Senate Office Bldg., 20510. (202) 224-4774

Representatives:

1. Pat Roberts, 1428 Longworth House Office Bldg., 20515. (202) 225-2715
2. Jim Jeffries, 128 Cannon House Office Bldg., 20515. (202) 225-6601
3. Larry Winn, Jr., 2416 Rayburn House Office Bldg., 20515. (202) 225-2865
4. Dan Glickman, 1507 Longworth House Office Bldg., 20515. (202) 225-6216
5. Robert Whittaker, 516 Cannon House Office Bldg., 20515. (202) 225-3911

When writing, the following form is appropriate:

Senators:

Honorable John Doe,
United States Senate
Address

Dear Senator Doe:

Representatives:

Honorable John Doe,
House of Representatives
Address

Dear Mr. Doe:

THE PRESIDENT

The White House,
1600 Pennsylvania Ave., N.W., 20500
(202) 456-1414

KANSAS LEGISLATURE

To write state Senators and Representatives, the following addresses may be used:

Senators:

Honorable John Doe
Senate Chambers
State Capitol Building
Topeka, KS 66612

Dear Senator Doe:

Phone: (913) 296-7300

Representatives:

Honorable John Doe
House of Representatives
State Capitol Building
Topeka, KS 66612

Dear Representative Doe:

(Phone: (913) 296-7500

THE GOVERNOR

State Capitol Bldg.
Topeka, KS 66612
(913) 296-3232

Medical School Codes

UNITED STATES

The following is a list of medical schools in the United States and Puerto Rico, existing and extinct, arranged in state order and showing the code number by which each school is designated in the geographical section of this Directory. Existing approved schools are listed in capital letters.

001 Alabama
001-02 UNIVERSITY OF ALABAMA SCHOOL OF MEDICINE, BIRMINGHAM

003 Arizona
003-01 UNIVERSITY OF ARIZONA COLLEGE OF MEDICINE, TUCSON

004 Arkansas
004-01 UNIVERSITY OF ARKANSAS SCHOOL OF MEDICINE, LITTLE ROCK

005 California
005-02 UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE, SAN FRANCISCO
005-06 UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE, LOS ANGELES
005-11 STANFORD UNIVERSITY SCHOOL OF MEDICINE, PALO ALTO
005-12 LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE, LOMA LINDA—LOS ANGELES

007 Colorado
007-02 UNIVERSITY OF COLORADO SCHOOL OF MEDICINE, DENVER

008 Connecticut
008-01 YALE UNIVERSITY SCHOOL OF MEDICINE, NEW HAVEN

010 District of Columbia
010-01 GEORGE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-02 GEORGETOWN UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-03 HOWARD UNIVERSITY COLLEGE OF MEDICINE, WASHINGTON

011 Florida
011-02 UNIVERSITY OF MIAMI SCHOOL OF MEDICINE, MIAMI
011-03 UNIVERSITY OF FLORIDA COLLEGE OF MEDICINE, GAINESVILLE

012 Georgia
012-01 MEDICAL COLLEGE OF GEORGIA, AUGUSTA
012-05 EMORY UNIVERSITY SCHOOL OF MEDICINE, ATLANTA

016 Illinois
016-01 RUSH MEDICAL COLLEGE, CHICAGO
016-02 UNIVERSITY OF CHICAGO PRITZKER SCHOOL OF MEDICINE, CHICAGO
016-04 The Hahnemann Medical College and Hospital, Chicago
016-06 NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, CHICAGO
016-11 UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE, CHICAGO
016-42 CHICAGO MEDICAL SCHOOL UNIVERSITY OF HEALTH SCIENCES, CHICAGO
016-43 LOYOLA UNIVERSITY STRITCH SCHOOL OF MEDICINE, MAYWOOD

017 Indiana
017-20 INDIANA UNIVERSITY SCHOOL OF MEDICINE, INDIANAPOLIS

018 Iowa
018-03 UNIVERSITY OF IOWA COLLEGE OF MEDICINE, IOWA CITY
018-75 College of Osteopathic Medicine and Surgery, Des Moines

019 Kansas
019-02 UNIVERSITY OF KANSAS SCHOOL OF MEDICINE, KANSAS CITY

020 Kentucky
020-02 UNIVERSITY OF LOUISVILLE SCHOOL OF MEDICINE, LOUISVILLE
020-12 UNIVERSITY OF KENTUCKY COLLEGE OF MEDICINE, LEXINGTON

021 Louisiana
021-01 TULANE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS
021-05 LOUISIANA STATE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS

022 Maine
022-01 Bowdoin Medical School, Brunswick-Portland

023 Maryland
023-01 UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE, BALTIMORE
023-07 JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE, BALTIMORE

024 Massachusetts
024-01 HARVARD MEDICAL SCHOOL, BOSTON
024-05 BOSTON UNIVERSITY SCHOOL OF MEDICINE, BOSTON
024-07 TUFTS UNIVERSITY SCHOOL OF MEDICINE, BOSTON

025 Michigan
025-01 UNIVERSITY OF MICHIGAN MEDICAL SCHOOL, ANN ARBOR
025-07 WAYNE STATE UNIVERSITY SCHOOL OF MEDICINE, DETROIT
025-12 MICHIGAN STATE UNIVERSITY COLLEGE OF HUMAN MEDICINE, EAST LANSING

026 Minnesota
026-04 UNIVERSITY OF MINNESOTA MEDICAL SCHOOL, MINNEAPOLIS

027 Mississippi
027-01 UNIVERSITY OF MISSISSIPPI SCHOOL OF MEDICINE, JACKSON

028 Missouri
028-02 WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-03 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, COLUMBIA
028-20 University Medical College of Kansas City
028-22 Ensworth Medical College, St. Joseph
028-34 ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-43 Kansas City College of Medicine and Surgery
028-46 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, KANSAS CITY
028-78 Kansas City College of Osteopathy & Surgery

030 Nebraska
030-05 UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE, OMAHA
030-06 CREIGHTON UNIVERSITY SCHOOL OF MEDICINE, OMAHA
030-07 Nebraska College of Medicine, Lincoln

033 New Jersey
033-05 COLLEGE OF MEDICINE & DENTISTRY OF NEW JERSEY — NEW JERSEY MEDICAL SCHOOL, NEWARK

034 New Mexico
034-01 UNIVERSITY OF NEW MEXICO SCHOOL OF MEDICINE, ALBUQUERQUE

035 New York
035-01 COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK
035-03 ALBANY MEDICAL COLLEGE OF UNION UNIVERSITY, ALBANY
035-06 STATE UNIVERSITY OF NEW YORK AT BUFFALO, SCHOOL OF MEDICINE, BUFFALO
035-08 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, BROOKLYN
035-09 NEW YORK MEDICAL COLLEGE, NEW YORK
035-10 Bellevue Hospital Medical College, New York
035-15 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, SYRACUSE

035-19 NEW YORK UNIVERSITY SCHOOL OF MEDICINE, NEW YORK
035-20 CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK
035-45 UNIVERSITY OF ROCHESTER SCHOOL OF MEDICINE AND DENTISTRY, ROCHESTER
035-46 ALBERT EINSTEIN COLLEGE OF MEDICINE OF YESHIVA UNIVERSITY, NEW YORK

036 North Carolina
036-01 UNIVERSITY OF NORTH CAROLINA SCHOOL OF MEDICINE, CHAPEL HILL
036-05 BOWMAN GRAY SCHOOL OF MEDICINE, WINSTON-SALEM
036-07 DUKE UNIVERSITY SCHOOL OF MEDICINE, DURHAM

038 Ohio
038-02 Eclectic Medical College, Cincinnati
038-06 CASE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE, CLEVELAND
038-40 OHIO STATE UNIVERSITY COLLEGE OF MEDICINE, COLUMBUS
038-41 UNIVERSITY OF CINCINNATI COLLEGE OF MEDICINE, CINCINNATI
038-43 MEDICAL COLLEGE OF OHIO AT TOLEDO, TOLEDO

039 Oklahoma
039-01 UNIVERSITY OF OKLAHOMA SCHOOL OF MEDICINE, OKLAHOMA CITY
039-79 OKLAHOMA COLLEGE OF OSTEOPATHIC MEDICINE AND SURGERY, TULSA

040 Oregon
040-02 UNIVERSITY OF OREGON MEDICAL SCHOOL, PORTLAND

041 Pennsylvania
041-01 UNIVERSITY OF PENNSYLVANIA SCHOOL OF MEDICINE, PHILADELPHIA
041-02 JEFFERSION MEDICAL COLLEGE OF THOMAS JEFFERSON UNIVERSITY, PHILADELPHIA
041-07 MEDICAL COLLEGE OF PENNSYLVANIA, PHILADELPHIA
041-09 HAHNEMANN MEDICAL COLLEGE AND HOSPITAL, PHILADELPHIA
041-12 UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE, PITTSBURGH
041-13 TEMPLE UNIVERSITY SCHOOL OF MEDICINE, PHILADELPHIA
041-77 Philadelphia College of Osteopathic Medicine, Philadelphia

042 Puerto Rico
042-01 UNIVERSITY OF PUERTO RICO SCHOOL OF MEDICINE, SAN JUAN

043 Rhode Island
043-01 BROWN UNIVERSITY DIVISION OF BIOLOGICAL AND MEDICAL SCIENCES, PROVIDENCE

045 South Carolina
045-01 MEDICAL UNIVERSITY OF SOUTH CAROLINA COLLEGE OF MEDICINE, CHARLESTON

047 Tennessee
047-05 VANDERBILT UNIVERSITY SCHOOL OF MEDICINE, NASHVILLE
047-06 UNIVERSITY OF TENNESSEE COLLEGE OF MEDICINE, MEMPHIS
047-07 MEHARRY MEDICAL COLLEGE SCHOOL OF MEDICINE, NASHVILLE

048 Texas
048-02 UNIVERSITY OF TEXAS MEDICAL BRANCH, GALVESTON
048-04 BAYLOR COLLEGE OF MEDICINE, HOUSTON
048-12 UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL, DALLAS
048-13 UNIVERSITY OF TEXAS MEDICAL SCHOOL, SAN ANTONIO

049 Utah
049-01 UNIVERSITY OF UTAH COLLEGE OF MEDICINE, SALT LAKE CITY

050 Vermont
050-02 UNIVERSITY OF VERMONT COLLEGE OF MEDICINE, BURLINGTON

051 Virginia
051-01 UNIVERSITY OF VIRGINIA SCHOOL OF MEDICINE, CHARLOTTESVILLE
051-04 MEDICAL COLLEGE OF VIRGINIA HEALTH SCIENCES DIVISION OF VIRGINIA COMMONWEALTH UNIVERSITY, RICHMOND

054 Washington
054-04 UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE, SEATTLE

055 West Virginia
055-01 WEST VIRGINIA UNIVERSITY SCHOOL OF MEDICINE, MORGANTOWN

056 Wisconsin
056-05 UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON
056-06 MEDICAL COLLEGE OF WISCONSIN, MILWAUKEE

FOREIGN MEDICAL SCHOOL CODES

CANADA

060 Alberta
060-01 University of Alberta Faculty of Medicine, Edmonton

061 British Columbia
061-01 University of British Columbia Faculty of Medicine, Vancouver

062 Manitoba
062-01 University of Manitoba Faculty of Medicine, Winnipeg

065 Ontario
065-01 University of Toronto Faculty of Medicine, Toronto
065-05 Queen's University Faculty of Medicine, Kingston

067 Quebec
067-01 McGill University Faculty of Medicine, Montreal

OTHER FOREIGN

118 Afghanistan
118-01 Faculty of Medicine, Kabul University, Kabul

132 Argentina
132-01 Facultad de Ciencias Medicas de la Universidad de Buenos Aires, Buenos Aires
132-02 Facultad de Ciencias Medicas de la Universidad Nacional de Cordoba, Cordoba
132-04 Facultad de Ciencias Medicas, Farmacia y Ramos Menores de la Universidad Nacional del Litoral, Rosario, Santa Fe
132-06 Facultad de Ciencias Medicas de la Universidad Nacional de Cuyo, Mendoza, Mendoza

143 Australia
143-03 Faculty of Medicine University of Sydney, Sydney, New South Wales

154 Austria
154-07 Medizinische Fakultat der Universitat Wien, Wien (407-26 from March 13, 1938 to June, 1945)

165 Belgium
165-01 Faculte de Medecine et de Pharmacie Universite libra de Bruxelles, Bruxelles

176 Bolivia
176-02 Facultad de Ciencias Medicas de la Universidad Mayor Real y Pontificia de San Francisco Xavier de Chuquisaca, Sucre
176-03 Facultad de Medicina de la Universidad Mayor de San Simon, Cochabamba

215 Cambodia
215-01 Ecole Royal de Medicine Du Cambode, Phnompenh

231 Chile
231-01 Facultad de Medicina de la Universidad de Chile, Santiago

242 China
242 China (also see 243 Effective January 1, 1977)
242-09 St. John's University (Pennsylvania Medical School, Shanghai, Kiangsu (Extinct)
242-16 National Shanghai Medical College, Shanghai, Kiangsu
242-17 West China Union University College of Medicine and Dentistry, Chengtu, Szechuan
242-22 Aurora University Faculty of Medicine, Shanghai, Kiangsu (Extinct)
242-39 Shansi University Medical College, Taiyuan, Shansi

243 China
243-38 National Honan University Medical College, Kaifeng, Honan (242-38 Prior to 1-1-71)
243-51 National Defense Medical Center, School of Medicine, Shanghai, Kiangsu (242-51 Prior to 1-1-71)

244 Taiwan
244 Taiwan (Formosa) effective 1-1-71
244-02 College of Medicine National Taiwan University, Taipai (385-02 Prior to 1-1-71)
244-04 Taipai Medical College, Taipai (385-04 Prior to 1-1-71)

264 Colombia
264-01 Facultad de Medicina de la Universidad Nacional de Colombia Cuidad Universitaria, Bogota, Cundinamarca
264-02 Faculdad de Medicina de la Universidad de Cartagena, Cartagena, Bolivar
264-04 Facultad de Medicina de la Pontificia Universidad Javeriana, Bogota, Cundinamarca
264-06 Facultad de Medicina de la Universidad de Caldas, Manizales, Caldas
264-07 Facultad de Medicina de la Universidad del Cauca, Popayan, Cauca

275 Cuba
275-01 Facultad de Medicina de la Universidad de la Habana, La Habana

308 Dominican Republic
308-01 Facultad de Medicina de la Universidad de Santo Domingo, Ciudad, Trujillo

319 Ecuador
319-01 Facultad de Ciencias Medicas de la Universidad Central, Quito

330 Egypt (United Arab Republic)
330-02 Kasr-el-Aini-Faculty of Medicine Cairo University, Cairo (Formerly Fouad First University Faculty of Medicine)
330-03 Faculty of Medicine Alexandria University, Alexandria

341 El Salvador
341-04 Facultad de Medicina Universidad Nacional del Salvador, San Salvador

352 England
352-05 School of Medicine University of Leeds, Leeds
352-07 University of London Faculty of Medicine, London
352-11 Registrable Qualifications granted by English Conjoint Board (Royal College of Surgeons of England/Royal College of Physicians of London)

385 Formosa (Taiwan)
385 (Also see 244 Taiwan [Effective 1-1-71])
385-01 Kaohsiung (takau) Medical College, Kaohsiung
385-02 College of Medicine National Taiwan University, Taipai
385-03 National Defense Medical Center, Taipai
385-05 China Medical College, Taichung

396 France
396-06 Faculte de Medecine de l'Universite de Paris, Paris, Seine
396-07 Faculte mixte de Medecine et de Pharmacie de l'Universite de Toulouse, Toulouse, Haute-Garonne

407 Germany

407 Also see 408-409—East and West Germany (Effective 1-1-71)
407-07 Medizinische Fakultat der Georg-August-Universitat, Gottingen, Niedersachsen
407-10 Medizinische Fakultat der Universitat Heidelberg, Heidelberg, Baden-Wurttemberg
407-15 Medizinische Fakultat der Phillipps-Universitat, Marburg/Lahn, Hessen
407-16 Medizinische Fakultat der Ludwig Maximiliams-Universitat, Munchen, Bayern
407-21 Medizinische Fakultat der Universitat Hamburg, Hamburg, Hamburg
407-23 Medizinische Fakultat der Johann-Wolfgang-Goethe-Universitat, Frankfurt-Am-Main, Hessen
407-33 Medizinische Fakultat der Freien Universitat Berlin, Berlin

409 Germany West

409-05 Medizinische Fakultat Albert-Ludwigs-Universitat Freiberg IM Breisgau
409-33 Medizinische Fakultat Freien Universitat, Berlin, Berlin (407-33 Prior to 1-1-71)

418 Greece

418-01 Faculty of Medicine National University of Athens, Athens
418-02 Faculty of Medicine University of Thessaloniki, Thessaloniki

451 Honduras

451-01 Facultad de Medicine y Cirugia de la Universidad Nacional Autonoma de Honduras, Tegucigalpa

473 Hungary

473-01 Orvosi Fakultas Tudomanyegyetem, Budapest

495 India (Goa)

495-01 University of Bombay, Affiliated Medical Colleges are:
a. Grant Medical College Bombay University, Bombay, Maharashtra
b. Seth Gorhandas Sunderdas Medical College Bombay University, Bombay, Maharashtra
495-04 Madras Medical College Madras University, Madras, Madras
495-08 Christian Medical College Punjab University, Ludhiana, Punjab
495-09 St. John's Medical College, Bangalore, Mysore (before June 1966: Government Medical College, Mysore University, Mysore)
495-11 Andhra Medical College Andhra University, Visakhapatnam, Andhra
495-16 Stanley Medical College Madras University, Madras, Madras
495-18 Assam Medical College Gauhati University, Dibrugarh, Assam
495-21 Osmania Medical College Osmania University, Hyderabad, Andhra
495-23 Medical College Baroda University, Baroda, Gujarat
495-27 Christian Medical College, Vellore, Madras
495-28 Byramjee Jeejeebhoy Medical College, Poona, Maharashtra
495-29 Government Medical College Punjab University, Patiala, Punjab
495-30 Sawai Man Singh Medical College Rajasthan University, Jaipur, Rajasthan
495-31 Medical College Kerala University, Trivandrum, Kerala
495-34 Gajra Rajo Medical College Vikram University, Gwalior Madhya Pradesh
495-35 Karnatak Medical College Karnatak University, Hubli, Mysore
495-36 All-India Institute of Medical Sciences, New Delhi, Delhi
495-37 Kasturba Medical College Karnatak University, Manipal, Mysore
495-41 G.S.V. Memorial Medical College Lucknow University, Kampur, Uttar Pradesh
495-47 Medical College Jabalpur University, Jabalpur, Madhya Pradesh
495-48 M.P. Shah Medical College Gujarat University, Jamnagar, Gujarat
495-49 Ghandhi Medical College Vikram University, Bhopai, Madhya Pradesh
495-50 Guntur Medical College Andhra University, Guntur, Andhra
495-54 Rajendra Medical College, Ranchi, Bihar
495-55 Sardar Patel Medical College, Bikaner, India
495-57 Kakatiya Medical College, Warangal, Andhra Pradesh
495-97 Dr. Vaishampayan Memorial Medical College, Shivaji University, Shalopur, Maharashitra

506 Indonesia
506-02 Faculty of Medicine Airlangga Airlangga University, Surabaya

517 Iran
507-01 Faculty of Medicine University of Teheran, Teheran

528 Iraq
528-01 Faculty of Medicine Baghdad University, Baghdad

539 Ireland
539-01 Faculty of Medicine Queen's University of Belfast, Belfast
539-02 National University of Ireland, Constitient Colleges are:
a. Faculty of Medicine University College, Dublin
b. Faculty of Medicine University College, Cork
c. Faculty of Medicine, Galway

550 Israel
550-01 The Hebrew University-Hadassah Medical School, Jerusalem

561 Italy
561-15 Facolta di Medicina e Chirurgia dell'Universita di Perugia, Perugia
561-19 Facolta di Medicina e Chirurgia dell'Universita di Siena, Siena

572 Japan
572-11 Tokyo Medical College (Nippon Ikadaigaku) Hongo, Tokyo (Extinct)
572-41 Faculty of Medicine Shinshu University, Matsumoto, Nagano

583 Korea (South)
583-01 Severence Medical College Yonsei University, Seoul
583-02 College of Medicine Seoul National University, Seoul
583-03 Korea University Medical College, Seoul
583-04 College of Medicine Kyong-Puk National University, Taegu
583-06 College of Medicine Chun Nam National University, Kwangiu
583-09 College of Medicine Pusan National University, Pusan

605 Lebanon
605-01 Medical School American University of Beirut, Beirut

627 Malta
627-01 Faculty of Medicine and Surgery Royal University of Malta, Valetta

649 Mexico
649-01 Facultad de Medicina de la Universidad Nacional Autonoma de Mexico, Mexico
649-02 Facultad de Medicina de la Universidad de Nuevo Leon, Monterrey, Nuevo Leon
649-06 Facultad de Medicina de la Universidad Nacional del Sureste, Merida, Yucatan
649-14 Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara Jalisco

660 Netherlands
660-61 Faculteit der Geneeskunde Universiteit Van Amsterdam, Amsterdam

671 New Zealand
671-01 Medical School University of Otago, Dunedin

704 Pakistan
704-01 King Edward Medical College, Lahore, West Pakistan
704-02 Dow Medical College, Karechi, Federal Capital Area
704-03 Dacca Medical College, Dacca, East Pakistan
704-04 Nishtar Medical College, Multan, West Pakistan
704-09 Khyber Medical College, Peshawar, North-West Frontier Province
704-10 Chittagong Medical College, Chittagong, East Pakistan (160-01 after 7-1-72)

726 Paraguay
726-01 Facultad de Medicina de la Universidad Nacional de Asuncion, Asuncion

737 Peru
737-01 Facultad de Medicina de San Fernando de la Universidad Nacional Mayor de San Marcos, Lima

737-05 Facultad de Medicina de la Universidad Nacional de San Agustin, Arequipa
737-06 Facultad de Medicina "Cayetano Heredia" de la Universidad Peruana de Ciencias Medicas y Biologicas, Lima

748 Phillipines
748-01 Faculty of Medicine and Surgery University of Santo Tomas, Manila
748-02 College of Medicine University of the Phillipines, Manila
748-07 College of Medicine Manila Central University, Manila
748-08 Institute of Medicine Far Eastern University, Manila
748-09 College of Medicine Southwestern University, Cebu City
748-10 College of Medicine University of the East, Quezon City
748-11 College of Medicine Cebu Institute of Technology, Cebu City

803 Scotland
803-01 Faculty of Medicine University of Aberdeen, Aberdeen
803-03 Faculty of Medicine University of Edinburgh, Edinburgh

836 South Africa
836-01 Medical School University of the Witwatersrand, Johannesburg

847 Spain
847-01 Facultad de Medicina de la Universidad de Barcelona, Barcelona
847-04 Facultad de Medicina de la Universidad de Madrid, Madrid
847-06 Faculdad de Medicina de la Universidad de Zaragoza, Zaragoza
847-08 Facultad de Medicina de la Universidad de Valencia, Valencia
847-10 Facultad de Medicina de la Universidad de Salamanca, Salamanca

869 Switzerland
869-01 Medizinische Fakultat der Universitat Basel, Basel
869-02 Medizinische Fakultat der Universitat Bern, Bern
869-05 Faculte de Medecine de l'Universite de Lausanne, Lausanne

875 Syria
875-01 Faculty of Medicine Damascus University, Damascus

Taiwan (See Formosa)

891 Thailand
891-01 Faculty of Medicine at Chulalongkorn Hospital University of Medical Sciences, Bangkok
891-02 Faculty of Medicine at Sariraj Hospital University of Medical Sciences, Thonburi

902 Turkey
902-01 Tip Fakultesi Istanbul Universitesi, Istanbul

913 Union of Soviet Socialist Republics
913-02 Voronez Medical Institute, Voronez

941 Viet-Nam South
941-01 Faculte mixte de Medicine et de Pharmacie Universite de Saigon, Saigon

957 Yugoslavia
957-02 Medicinski Fakultet Univerziteta u Beogradu, Beograd

IS YOUR LISTING ACCURATE?

If there is an error in your listing, please notify the Executive Office so we can correct our records.

Send corrections to:

Ramona Perez, Membership Secretary
The Kansas Medical Society
1300 Topeka Avenue
Topeka, KS 66612

Alphabetical Listing

A

ABBAS, DILAWER H, WICHITA
ABBUEHL, DON R, CHANUTE
ABDOU, NABIH I, KANSAS CITY
*ACEVEDO, ALFREDO, WICHITA
ADAMS, DWIGHT, OSAGE CITY
ADAMS JR, MARCUS W, HUTCHINSON
AGAN, LAWRENCE M, TOPEKA
AGUSTIN, CONRADO M, WICHITA
AHLSTRAND, RICHARD A, WICHITA
AILLON, ALEJANDRO J, HALSTEAD
AKERS, GUY I, FORT SCOTT
AL-BAGHAL, MOHAMMAD, LEAVENWORTH
ALBRIGHT, JEROLD D, HUTCHINSON
ALDERSON, THOMAS W, GREAT BEND
ALDIS, HENRY, FORT SCOTT
ALDIS, WILLIAM, FORT SCOTT
*ALDOROTY, NEIL, WICHITA
ALEXANDER, CHARLES E, KANSAS CITY
ALEXANDER, CLYDE W, KANSAS CITY
ALEXANDER, ELIZABETH, WICHITA
ALFONSO, MANJEL, WICHITA
ALGIE, WILLIAM H, KANSAS CITY
ALLBRITTEN JR, FRANK F, CUNNINGHAM
ALLEGRE, ANN, KANSAS CITY
ALLEN, FRANCES A, NEWTON
ALLEN, JAMES E, HAYS
ALLEN, MARK LYNN, KANSAS CITY,MO
ALLEN, MAX S, SHAWNEE MISSION
ALLEN, MONTE L, SALINA
*ALLEN, PHILLIP M, WICHITA
ALLEN, RAY E, LIBERAL
ALLEN, TIMOTHY E, KANSAS CITY
ALLEN, WILLIAM R, KANSAS CITY
ALLEN JR, WILLIAM R, SHAWNEE MISSION
ALMONTE, PRISCILLA C, WICHITA
ALMONTE, RODOLFO O, WICHITA
ALQUIST, VERYL D, BAXTER SPRINGS
ALTENBERND, ELVIN CONRAD, SHAWNEE MISSION
ALVAREZ, LUIS A, SHAWNEE MISSION
ALVAREZ, NORBERTO, ARKANSAS CITY
AMARE, MAMMO, KANSAS CITY
AMAWI, MOHAMMAD S, DODGE CITY
AMBLER, CARL D, PRATT
AMEND, DOUGLAS J, EMPORIA
*AMMAR, ALEX D, WICHITA
ANDERSON, DALE W, AUGUSTA
ANDERSON, EUGENE G, WICHITA
ANDERSON, JODY, SALINA
ANDERSON, LARRY R, WELLINGTON
ANDERSON, LYLE B, GREAT BEND
ANDERSON, SEVERT A, CLAY CENTER
ANDERSON, WINSTAN L, LAWRENCE
ANDERSON JR, HERMAN L, LEAVENWORTH
ANTRIM, PHILIP JENIFER, ANTHONY
ANWAR, M ZIA, LEAVENWORTH
APPENFELLER, WILLIAM O, OSAWATOMIE
APPLEGATE JR, FRANCIS R, HAYS
ARAKAWA, KASUMI, KANSAS CITY
ARENAL, ANGELA C, KANSAS CITY
ARGO, DONALD A, MARYSVILLE
ARGOSINO, RODOLFO, WICHITA
ARMBRUSTER, ALBERT A, SHAWNEE MISSION
ARMSTRONG, HAROLD J, PITTSBURG
ARONOFF, MICHAEL E, OLATHE
ARREDONDO, MARIO, TOPEKA
ARROYO, ZEFERINO, GARDEN CITY
ARTMAN, JOHN C, HAYS
ARTZ, TYRONE D, WICHITA
ARTZER, DENNIS C, TOPEKA
ARUNAKUL, PUNYA, TOPEKA
ASHER, MARC A, KANSAS CITY
ASHKAR, ADNAN A, LEAVENWORTH
ASHLEY, BYRON J, TOPEKA
ASHLEY, SAMUEL G, CHANUTE
ASHLEY JR, B JOHN, TOPEKA
ASHMORE, ARTHUR L, WICHITA
ATHON, MERRILL O, SHAWNEE MISSION
ATKIN, JOHN D, YATES CENTER
ATKINS JR, FLOYD L, KANSAS CITY
ATLURU, NARAYANA RAO, TOPEKA
ATWOOD, M DALE, KINSLEY
AUCAR, ALFREDO, ARKANSAS CITY
AUCHARD, VIRGIL M, LAWRENCE
AUNINS, JOHN, WICHITA
AUSTENFELD, MARK S, WICHITA
AUSTIN, JOHN O, GARDEN CITY
AUSTIN, KENNETH O, GOODLAND
AVERILL, STUART C, TOPEKA
AVES, AGNES, PARSONS
AVES, RENATO B, PARSONS
AVILA, OSCAR A, DODGE CITY
AYUTHIA, ISSARA I, DODGE CITY

B

BACANI, OSWALDO, FREDONIA
BACON, ARTHUR H, LAKE WORTH,FL
BADEEN II, LOUIS JOHN, SHAWNEE MISSION
BAEHR, RALPH H, TOPEKA
BAEKE, JOHN O, SHAWNEE MISSION
BAILEY, COLIN, HALSTEAD
BAILEY, DONALD C, WICHITA
BAILEY, WILLIAM A, LAWRENCE
BAILIE, MICHAEL DAVID, KANSAS CITY
BAIR, ALBERT E, INDEPENDENCE
BAIR, GLENN O, TOPEKA
BAKER, FREDERICK C, TOPEKA
BAKER, HENRY K, CHANUTE
BAKER, PHILLIP L, TOPEKA
BAKER, RAY D, TOPEKA
BAKER, RICHARD B, MANHATTAN
BAKER, WILLIAM STEVEN, SHAWNEE MISSION
BAKER DO, ROBERT R, ARKANSAS CITY
BALANOFF, ARNOLD Z, SHAWNEE MISSION
BALL, RALPH G, MANHATTAN
BANKS, GILBERT, COFFEYVILLE
BANKS, ROBERT E, PAOLA
BANSAL, SATISH C, SHAWNEE MISSION
BAPTIST, JEREMY E, SHAWNEE MISSION
BARABAN, MARC R, TOPEKA
BARBA, ESTRELLA G, WICHITA
BARBA JR MD, ANTONIO P, WICHITA
BARBER, JAMES L, AUGUSTA
BARBERA, PORTER E, INDEPENDENCE
BARE II, CHARLES E, SHAWNEE MISSION
BARKER, BENJAMIN W, WICHITA
BARKER, ELIZABETH B, SHAWNEE MISSION
BARKER, JAMES BERTON, SHAWNEE MISSION
BARKER, PATRICK N, PRATT
BARKER, ROYAL A, COUNCIL GROVE
BARKER, STEVEN E, MINNEAPOLIS
BARLOW, JOHN M, MANHATTAN
BARNARD III, JAMES A, GARDEN CITY
BARNES, MARIAN, LANCASTER,CA
BARNETT, ARNOLD M, WICHITA
BARNETT JR, THOMAS E, SHAWNEE MISSION
BARNHART, RONALD J, SHAWNEE MISSION
BARNHILL, C ALTON, TOPEKA
BARNHORST, DONALD A, SHAWNEE MISSION
BARR, RICHARD N, SHAWNEE MISSION
BARRICK, BRUCE, SHAWNEE MISSION
BARRY, DAVID R, LEAVENWORTH
*BARTAL, ELY, WICHITA
BARTLETT, WAYNE C, WICHITA
BASCOM, CHARLES H, MANHATTAN
BASCOM, GEORGE S, MANHATTAN
BASER, ALI N, TRAVIS AFB,CA
BASHAM, JAMES J, FORT SCOTT
BASINGER, BRADLEY B, KANSAS CITY
BASS II, ORAL E, WICHITA
BASS JR, LEWIS N, KANSAS CITY
BASSETT, P MARCUS, HIAWATHA
BATES, MICHAEL D, WICHITA
BATES, MICHAEL NICHOLS, NEWTON
BATNITZKY, SOLOMON, KANSAS CITY
BATTISTE, CYNTHIA ELAINE, WICHITA
BATTY, LARRY H, SHAWNEE MISSION
BATTY, THOMAS V, SHAWNEE MISSION
BAUCOM, KARAN YVONNE, TOPEKA
BAUCOM-COPELAND, SHARON LAVARNE, TOPEKA
BAUDE, EUGENE L ANDRE, TOPEKA
BAUER, MARTIN L, SHAWNEE MISSION
BAUER, THOMAS A, HUTCHINSON
BAUM, ARNOLD H, DODGE CITY
BAUMAN, M LEON, WICHITA
BAUMANN, PAUL A, WICHITA
BAXTER, KIRKMAN G, SHAWNEE MISSION
BAXTER, W REESE, SALINA
BAYLES, HUGH G, FREDONIA
BEACH, RICHARD R, TOPEKA
BEAHM, ANOL W, GREAT BEND
BEAHM, DONALD E, GREAT BEND
BEAHM, EDGAR H, INDEPENDENCE
BEAL, RAYMOND J, FREDONIA
BEALE, DAVID A, TOPEKA
BEAMON, RICHARD F, SHAWNEE MISSION
BEATY, JAMES R, TOPEKA
BEAVER, JAMES L, WICHITA
BEBAK, DONALD M, WICHITA
*BECK, CHARLES W, WICHITA
BECK, JOSEPH D, TOPEKA
BECKER, KARL E, WICHITA
BECKER, LESLIE E, KANSAS CITY
BEDFORD, D R, TOPEKA
BEEBE, EDMER, OLATHE
BEELMAN, FLOYD C, TOPEKA
BEGGS, DAVID F, GARDEN CITY
BEHRHORST, CARROLL D, GUATEMALA
BEIDERWELL, PAUL L, BELLEVILLE
BELCHER, GEORGE D, COLUMBUS
BELL, DELORIS W, SHAWNEE MISSION
BELL, MARGARET E, APO NEW YORK,NY
BELLAR, RALPH E, HARPER
BELLER, WILLIS L, TOPEKA
BELOT JR, MONTI L, LAWRENCE
BELZER, EDWARD G, SHAWNEE MISSION
BENA, JAMES H, PITTSBURG
BENAGE, JOHN F, FORT SCOTT
BENNETT, CHARLES A, LEAVENWORTH
BENTON, JAY S, NEWTON
BENZ, LAURIE J, WICHITA
BERGIN, JAMES J, KANSAS CITY
BERKEY, VERNON A, PITTSBURG
BERKLEY, DON H, ABILENE
BERKLEY, NORMAN W, SENECA
BERLAND, DAVID I, TOPEKA
BERNER, NEAL E, WAKEENEY
BERRY, JOHN M, SHAWNEE MISSION
BEST, JOHN F, EMPORIA
BETHEL, CHANDLER S, WICHITA
BETTERTON, DALE C, DODGE CITY
BEUGELSDIJK, HENRY PETER, HALSTEAD
BHARGAVA, ASHOK KUMAR, LACROSSE
BHARGAVA, BAIKUNTH N, WICHITA
BHARGAVA, SHOBHANA, LA CROSSE
BICHLMEIER, FRANKLIN G, KANSAS CITY
BIERI, PETER V, LAWRENCE
BIERLEIN, KENNETH J, PITTSBURG
BIERMANN, HENRY J, WICHITA
BIERMANN, WILLIAM J, WICHITA
BIGGS, DENNIS, ABILENE
BIGLER, F CALVIN, GARDEN CITY
BIGONGIARI, LAWRENCE R, KANSAS CITY
BIKALES, VICTOR WILLIAM, SHAWNEE MISSION
BILLINGS, THOMAS, MCPHERSON
BILLINGSLEY, THAO H, KANSAS CITY
BINGAMAN, ROBERT W, WICHITA
BINYON, KERNIE W, WICHITA
BISHOP, FRANCIS E, SHAWNEE MISSION
BISHOP, RODNEY LEE, LAWRENCE
BITTENBENDER, LEE R, LAWRENCE
BITZER, DONALD A, WASHINGTON
BIXLER II, THOMAS J, KANSAS CITY
BLACK, CYRIL V, PRATT
BLACK, WILLIAM L, APO NY,NY
BLACKBURN, ROBERT W, COUNCIL GROVE
BLAIR, T RICHARD, LAWRENCE
BLAKE, HENRY S, TOPEKA
BLANK, JOHN N, HUTCHINSON
BLANKENSHIP, JIM D, OTTAWA
BLAYLOCK, HOYT C, WICHITA
BLETZ, DONALD B, SHAWNEE MISSION
BLISS, JOY V, OLATHE
BLOOD, MARY J, WICHITA
BLOOM, L THEIL, KINGMAN
BLOOM, RODNEY LAMONT, WICHITA
BLOXHAM, THOMAS J, WICHITA
BLUM DO, MICHAEL A, OLATHE
BODENSTEINER, DAVID C, KANSAS CITY
BOESE, KENNETH M, MANHATTAN
BOGGAN, MICHAEL D, KANSAS CITY
BOLES, J MICHAEL, SHAWNEE MISSION
BOLES, R DALE, DODGE CITY
BOLINGER, ROBERT E, KANSAS CITY
BOLLMAN, CHARLES S, JUNCTION CITY
BOLT, MICHAEL, PARSONS
BOLTON, VICTOR E, KANSAS CITY
BOND, ROGER C, WICHITA
BONEBRAKE, C RICHARD, TOPEKA
BOREL, DAVID, TOPEKA
BORGENDALE, LLEWELLYN V, WAMEGO
BORRA, MARIO J, HUTCHINSON
BOS, NORMAN C, HUTCHINSON
BOSILEVAC, FRED N, KANSAS CITY
BOSILJEVAC JR, JOSEPH E, EMPORIA
BOSSE, FRANK K, ATCHISON
BOSWELL, H CRAIG, SHAWNEE MISSION
BOUDA, DAVID W, HUTCHINSON
BOUDET, ROBERT A, KANSAS CITY,MO
BOUDREAUX, VELTIN J, HALSTEAD
BOWEN, CLOVIS W, TOPEKA
BOWEN JR, HARRY J, TOPEKA
BOYD, SPENCER H, TOPEKA
BOYD, Z REX, WICHITA
BOYDEN, MARY S, LAWRENCE
BOYER, ROBERT E, KINGMAN
BOYLE, HUGH H, WICHITA
BRACKETT JR, CHARLES E, KANSAS CITY
BRADA, DONALD ROBERT, HUTCHINSON
BRADEN, BILL L, WAMEGO
BRADLEY, H RUSSELL, EMPORIA
BRADLEY, J RODERICK, GREENSBURG
BRADY, CHARLES S, ATCHISON
*BRAHMAN, HERBERT D, TOPEKA
BRAKE, DAVID, WICHITA
BRANDSTED, ERNEST C, MCPHERSON
BRANSON, VERNON L, LAWRENCE
BRAUN, EDWARD W, FORT SCOTT
BRAUN, KENNETH, WICHITA
BRAUN, ROBERT W, TOPEKA
BRAUN, THOMAS G, WICHITA
BRAUN, WILLIAM T, PORT ORANGE,FL
BRAUN III, WILLIAM T, WICHITA
BRAUNSDORF, ROBERT L, TOPEKA
BRAVERMAN, DAVID ELLIOTT, SHAWNEE MISSION
BRAY, AVIS PAGE, CONCORDIA
BRECKBILL, DAVID L, WICHITA
BRENNER, WILLIAM R, LARNED
BRETHOUR, LESLIE J, JUNCTION CITY
BREWER, MARSHALL A, ULYSSES
BRIAN, ROBERT M, EL DORADO
BRIBACH, EUGENE J, ATCHISON
BRIDGENS, JAMES G, SHAWNEE MISSION
BRIDWELL, RUSSELL E, TOPEKA
BRILLHART, MAXINE T, KANSAS CITY
BRINTON, E HOLMES, WICHITA
BRINTON, EDWARD S, WICHITA
BRITO, RAUL E, WICHITA
BROCKHOUSE, JOHN P, EMPORIA
BROOKER, ROBERT M, SANTA BARBARA,CA
BROOKS, WILLIAM HENRY, KANSAS CITY
BROSIUS, FRANK C, WICHITA
BROTHERS, MARY ELIZABETH, KANSAS CITY,MO
BROUCEK, FRANCIS J, SHAWNEE MISSION
BROWN, ALEX L, CHAUTAUQUA,NY

* Probationary members.

BROWN, C EVERETT, STAFFORD
BROWN, C REIFF, GREAT BEND
BROWN, DAVID J, WICHITA
BROWN, FRED E, ST MARYS
BROWN, JOHN V, KANSAS CITY
*BROWN, MICHAEL P, WICHITA
BROWN, PAUL W, OLATHE
BROWN, ROBERT L, WICHITA
BROWN, ROBERT M, MANHATTAN
BROWN, ROBERT O, ATCHISON
BROWN, ROBERT WAYNE, SALINA
BROWN, RONALD C, WICHITA
BROWN, RONALD L, WICHITA
BROWN, VAL J, WICHITA
BROWN, WILLIAM R, SHAWNEE MISSION
BROWN-SANDERS, CAROLINE, LEES SUMMIT,MO
BROWNING, JIMMIE L, COTTONWOOD FALL
BROWNING, WILLIAM H, WICHITA
BROWNING, WILLIAM R, MADISON
BROWNRIGG, RICHARD L, DODGE CITY
BROKTERMAN, STEVEN JOSEPH, SHAWNEE MISSION
BRUMMETT, RICHARD R, SALINA
BRUNER, STEVEN C, LAWRENCE
BRUNER JR, KENNETH W, WICHITA
BRUNFELDT, JOAN KRAUS, LAWRENCE
BRUNGARDT, BERNARD A, SALINA
BRUNING, ROGER MARION, SHAWNEE MISSION
BRUNO, JAMES W, GARDEN CITY
BRYAN, EMERY C, ERIE
BUBB, STEPHEN K, KANSAS CITY
BUBECK, RALPH W, WICHITA
BUCK JR, BEN H, WICHITA
BUCK JR, HENRY W, LAWRENCE
BUCKMAN, MARTIN SPALDING, SHAWNEE MISSION
BUCKRIDAN, LATIF, WICHITA
BUDETTI, JOSEPH A, HOLLYWOOD,FL
BUESING, OLIVER R, LEAVENWORTH
BULA, RALPH E, HAYS
BUNKER JR, HERBERT L, JUNCTION CITY
BURDICK, BRUCE M, MANHATTAN
BURDZIK, EBERHARD G, TOPEKA
BURGER, J DALE, HUTCHINSON
BURGER, PAUL B, SHAWNEE MISSION
BURGER, WILLIAM E, KANSAS CITY
BURGESON, FRANK G, EMPORIA
BURGESS, ARTHUR P, OSWEGO
BURKE, JAMES J, FORT SCOTT
BURKE, JOSEPH V, ATCHISON
BURKET JR, GEORGE E, KINGMAN
BURKMAN, REUBEN J, CHANUTE
BURNETT, A DEAN, HALSTEAD
BURNEY, WILLIAM W, WICHITA
BURNEY II, WILLIAM W, WICHITA
BURPEE, JAMES F, WICHITA
BURTON, JERALD ALBERT, SHAWNEE MISSION
BUSCH, ANTHONY B, DODGE CITY
BUSTOS, JONAS G, HERINGTON
BUTCHER, THOMAS P, EMPORIA
BUTH, DENNIS K, WICHITA
BUTIN, J WALKER, WICHITA
BUTIN, RICHARD E, SHAWNEE MISSION
BUTLER, DORIS C, WICHITA
BUTT, MUHAMMAD, CONCORDIA
BUTTERFIELD, MARI ANNE, KANSAS CITY
BYLANDER DO, TERESA I, TOPEKA
BYRNE, JAMES PERRY, WICHITA

C

CABLE, THOMAS M, WINCHESTER,KY
CABRERA, ALBERTO, ELKHART
*CACHIA, RICHARD M, TOPEKA
CAEDO, CARMELITA D, LIBERAL
CAIN, IVAN W, SHAWNEE MISSION
CALBECK, JOHN, GARDEN CITY
CALDERON, JAIME, KANSAS CITY
CALIENDO JR, DANIEL J, WICHITA
CALKINS, JOHN W, SHAWNEE MISSION
CALKINS, LARRY L, SHAWNEE MISSION
CALKINS, W GRAHAM, KANSAS CITY,MO
CAMERON, WILLIAM J, KANSAS CITY
CAMPBELL, EDWARD G, EMPORIA
CAMPBELL, FRANCES S, NEWTON
CAMPBELL, GARLAND L, ARKANSAS CITY
CAMPBELL, WILLIAM H, COFFEYVILLE
CAMPION, WOODROW M, LIBERAL
*CANNON, MICHAEL W, WICHITA
CAPPER, STANLEY L, WICHITA
CARDUFF, JAY J, SHAWNEE MISSION
CAREY, LARRY J, PARSONS
CARLETON, RICHARD C, CLAY CENTER
CARLIN, JAMES WILLARD, KANSAS CITY
CARLSON, EARL V, HAYS
CARLSON, TERRY S, WICHITA
CARNAHAN, ROBERT L, LAWRENCE
CARNEY, MYRTLE S, FT WORTH,TX
CARPENTER, PAUL R, KANSAS CITY
CARPER, IVAN H, NEWTON
CARPER, OWEN E, NEWTON
CARRASCO, LENOR C, SHAWNEE MISSION
CARREAU, ERNEST P, GEORIDGE,CO
CARRO, F AURELIO, WINFIELD
CARRO, TONY L, SHAWNEE MISSION
*CARSON DO, BERTRAM H, WICHITA
CARTER, MACK A, WICHITA
CARVER, LARRY A, TOPEKA
CASADY, GILBERT N, HUTCHINSON
CASEY, JAMES, HUTCHINSON
CASEY, JOHN J, WICHITA
CASHMAN JR, MAURICE R, TOPEKA
CASTEEL, CHARLES K, SHAWNEE MISSION
*CASTRO, JUAN A, TOPEKA
CATHCART-RAKE, WILLIAM F, SALINA
CATHEY, ROBERT H, MANHATTAN
CATTANEO, ERNEST A, SHAWNEE MISSION
CAUBLE, WILBUR G, WICHITA
CAVANAUGH, CLAIR J, GREAT BEND
CAVANAUGH, JOHN W, TOPEKA
CAVITT, ROBERT F, SHAWNEE MISSION
CAWLEY, LEO P, WICHITA
CECIL III, JOHN, HAYS
CEDERLIND, CRANSTON JAY, SHAWNEE MISSION
CENAC, MARK T, LEAVENWORTH
CHAFFEE, DEAN C, ABILENE
CHAFFIN JR, GOODLOE S, PRATT
CHALIAN, ALEXANDER R, KANSAS CITY
CHAMBERLIN JR, CECIL R, TOPEKA
CHANEY, ERNIE J, BELLEVILLE
CHANG, C H JOSEPH, KANSAS CITY
CHANG, FONG CHUNG, TOPEKA
CHANG, FREDERIC C, WICHITA
CHANG, SHU FANG, SHAWNEE MISSION
CHAPMAN, JAMES H, WICHITA
CHAPPUIE, WILLIAM G, INDEPENDENCE
CHARO, FREDERICK H, WICHITA
CHAVALA, SUDARSAN, FT LEAVENWORTH
CHEDIAK, ELIAS, LAWRENCE
CHEN, CHU-CHI, TOPEKA
CHEN, TAK-MING, TOPEKA
CHENOWETH, JOHN R, D.O., OLATHE
CHERRY JR, ARTHUR C, TOPEKA
CHERVEN, PHILIP L, HUTCHINSON
*CHI, IL-SUNG, WICHITA
CHIN, TOM D, KANSAS CITY
CHO, CHENG T, KANSAS CITY
CHO, SECHIN, WICHITA
CHONKO, ARNOLD M, KANSAS CITY
*CHOPRA, RAMAN, WICHITA
CHOTIMONGKOL, ANUPONG, DODGE CITY
CHOW, STANLEY Y, FORT SCOTT
CHOY, JAMES K L, SUN CITY,AZ
CHRISTENSEN, MARION D, KIOWA
CHRISTIAN, STANLEY J, SHAWNEE MISSION
CHRISTMAN JR, CARL, WICHITA
CHRONISTER, BERT, NEODESHA
CHRYSANT, STEVEN G, KANSAS CITY
CHUBB, RICHARD M, BAXTER SPRINGS
CHUN, CHUNG S, KANSAS CITY
CHUNG, JOHN J, SHARON SPRINGS
CISKEY, WILLIAM J, EUREKA
CLAASSEN, MILTON A, NEWTON
CLARK, COURTNEY, WICHITA
CLARK, CRAIG N, TOPEKA
CLARK, DAVID H, SALINA
CLARK, LAURENCE A, WAMEGO
CLARK, ORVILLE R, ST PETERSBURG,FL
CLARK, RAY A, LAKE CHAS,LA
CLARK, ROBERT THOMAS, GUATEMALA CEN A,
CLEAVER, EDGAR M, WICHITA
CLENDENIN, ROBERT KEELE, OLATHE
CLIFTON, H DAVID, WICHITA
*CLINE, BYRON W, WICHITA
CLINTON, DALE L, LAWRENCE
CLYMER, DAVID V, SHAWNEE MISSION
COALE, LLOYD H, KANSAS CITY
COBB, LESLIE H, MULVANE
COCHRAN, PAUL W, TOPEKA
CODY, DOROTHY, HAYS
CODY, JOHN, HAYS
COE, RICHARD D, SHAWNEE MISSION
COFFEY, ROY B, SALINA
COHEN, JUSTIN THOMAS, WICHITA
COHEN, LOUIS, TOPEKA
COHEN, ROBERT A, SHAWNEE MISSION
COHLMIA, JERRY B, WICHITA
COHN, STEVEN G, SHAWNEE MISSION
COHNBERG, ROSELLEN E, CEDAR VALE
COKELEY, JOHN M, TOPEKA
*COKER, W LAURENCE, TOPEKA
COLDSMITH, DONALD C, EMPORIA
COLE, WARD M, WELLINGTON
COLEMAN, GARY, ABILENE
COLEMAN, THOMAS J, WICHITA
COLIP, F MERLYNN, NORTON
COLLIER, HAROLD W, WICHITA
COLLIER, WILLIAM J, MCPHERSON
COLLINS, DEAN T, TOPEKA
COLLINS, EDWARD JOSEPH, TOPEKA
COLLINS, ELISABETH B, TOPEKA
COLLINS, FRANCIS T, TOPEKA
COLLINS, SHARON A, GARDEN CITY
COMBS, G RALPH, LEAVENWORTH
COMBS, PETER S, LEAVENWORTH
CONARD, CLAIR C, DODGE CITY
CONCEPCION JR, EUGENIO S, WICHITA
CONNELLY, JOHN C, TOPEKA
CONNELLY, MAURICE R, SALINA
CONNER, BRIAN, SALINA
CONRADY, PETER A, WICHITA
CONROY, ROBERT M, TOPEKA
COOK, DONALD RAY, WICHITA
COOK, G EDWARD, WICHITA
COOK, JAMES D, KANSAS CITY
COOKE, ALLAN R, KANSAS CITY
COOLEY, DAVID A, SHAWNEE MISSION
COOLEY, DENNIS M, TOPEKA
COOMER, TYLER E, PITTSBURG
COONFIELD, JAMES W, KANSAS CITY
COOPER, ARTHUR E, NORTON
COOPER, JACK R, SHAWNEE MISSION
COOPER, KENT J, PITTSBURG
COOPER, LEO F, SHAWNEE MISSION
*COOPER, MICHAEL K, WICHITA
COPELAND, GARY A, JUNCTION CITY
COPENING, TELL B, IOLA
CORBIN, MURRAY D, SHAWNEE MISSION
CORDER, ROBERT L, HIGHLAND
CORDER, S SCOTT, OTTAWA
CORONADO, EFRAIN, GARDEN CITY
COSSETTE, JERROLD E, SALINA
COSSMAN, F PRICE, WICHITA
COTTON, ROBERT T, TOPEKA
COULTER, HENRY F, SHAWNEE MISSION
COULTER, THOMAS B, SHAWNEE MISSION
COULTER DO, THAYNE A, CONCORDIA
COVERT, THOMAS J, SALINA
COWLES, GORDON T, WICHITA
COX, ROBERT H, HAYS
COX III, IRA L, KANSAS CITY
COX JR, IRA, SHAWNEE MISSION
COX JR, WALLACE F, KANSAS CITY
COYLE, JOHN F, COFFEYVILLE
CRAIG, CHARLES C, NEWTON
CRAIG, THOMAS A, JUNCTION CITY
CRAM, ERNEST R, ST FRANCIS
CRAM JR, OLE R, LARNED
CRAMER, GUY W, PARSONS
CRAMM, RUSSELL E, HAYS
CRANE, C HERBERT, MANHATTAN
CRANE, DAVID D, WICHITA
CRANSTON, STEPHEN D, NEWTON
CRARY, JOHN E, TOPEKA
CROCKETT, CHARLES A, KANSAS CITY
CRONEMEYER, RICHARD L, KANSAS CITY,MO
CRONIN, DONALD J, WICHITA
CROUCH, STEVEN W, TOPEKA
CROUCH, WILLIAM H, TOPEKA
CROW, ERNEST W, WICHITA
CROWLEY, EDWARD K, WICHITA
CULP, LOUIS M, KANSAS CITY
CULTRON, FRANK T, SALINA
CULVER, WARREN T, LAWRENCE
CUMMINGS, RICHARD J, WICHITA
CURRAN, KEVIN E, SHAWNEE MISSION
CURTIS, MIRJANA R, KANSAS CITY

D

D'SOUZA, BISMARCK C, SALINA
DAEHNKE, SIGURD S, WINFIELD
DAHL, ASHER W, COLBY
DAHL, DENNIS R, LAWRENCE
DAILEY, RONALD F, JOHNSON
DAIZ, ANTONIO S, PARSONS
*DAKHIL, SHAKER R, WICHITA
DALUM, PETER JOSEPH, CLAY CENTER
DANIELS, ROBERT M, WICHITA
DANIELS III, H BRUCE, WICHITA
DARR, RICHARD B, KANSAS CITY
*DARRAH, CAROL J N, WICHITA
DAVIDSON, HARRY T, WICHITA
DAVIS, CHESTER R, TOPEKA
DAVIS, CHRISTOPHER G, KANSAS CITY
DAVIS, DAVID H, LARNED
DAVIS, DAVID R, EMPORIA
DAVIS, GEORGE R, ELLSWORTH
DAVIS, PAUL H, WICHITA
DAVIS, RICHARD E, SHAWNEE MISSION
DAVIS, RONALD B, WICHITA
DAVIS JR, JAMES W, KANSAS CITY,MO
DAY, HOWARD, WICHITA
DAY, HUGHES W, KANSAS CITY
DE BAKKER, JAN B, WICHITA
*DE HART, ARTHUR DONIVA, WICHITA
DE SMET, ARTHUR AUGUST, KANSAS CITY
DE SOUZA, DERRICK J, LEAVENWORTH
DECHAIRD, THOMAS, WESTMORELAND
DECKER, DONALD D, HALSTEAD
DEGNER, JAMES B, GREAT BEND
DEITZ, MICHAEL R, KANSAS CITY
DEJONG, DAVID C, WICHITA
DELCORE, ROMANO, TOPEKA
DELGADO, SERGIO, TOPEKA
DELLETT, KENNETH B, EL DORADO
DELP, MAHLON H, SHAWNEE MISSION
DELPHIA, ROBERT E, OLATHE
DEMOSS, ELEANOR P, WICHITA
DEMOTT, WAYNE R, KANSAS CITY
DENISON, TERRY R, SHAWNEE MISSION
DEPENBUSCH, FRANCIS L, HUTCHINSON
DEPOE, JOSEPH H, WICHITA
DERRINGTON, KENNETH L, SHAWNEE MISSION
DESOIGNIE, RAFAEL R, TOPEKA
DETAR, GEORGE F, IOLA
DIACON, JAMES L, WICHITA
DIALLO, GASTON I, KANSAS CITY
DICK, ARTHUR R, KANSAS CITY
DICK, WILLIS G, IOLA
DICK JR, HENRY J, CHANUTE
DICKERSON, WILLIAM E, HONOLULU,HI
DICKERSON II, W JOHN, SALINA
DICKINSON, CHARLES R, COFFEYVILLE
DIEDERICH, DENNIS A, KANSAS CITY
DIEHL, ANTONI M, KANSAS CITY,MO
DIENER, CLAYTON H, HESSTON
DILL, ROONEY, ATWOOD
DILLON, WILLIAM L, PARSONS

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DONAHDE, ODRVAL H, MEADE
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ODNNELL, JAMES M, WICHITA
DONNELLY, F MICHAEL, KANSAS CITY
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DOUGHERTY, THOMAS M, GARNETT
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DOWNING, GREGDRY, WICHITA
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DREVETS, CURTIS C, WICHITA
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DUCKETT II, THOMAS G, SHAWNEE MISSION
DUICK, GREGDRY, WICHITA
DUJOVNE, CARLDS A, KANSAS CITY
DUNAGIN, JACK A, TOPEKA
*DUNIVEN, PHILIP L, TOPEKA
DUNLAP, RICHARD L, LAWRENCE
DUNN, DANIEL R, SCDTT CITY
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DUNN, MARVIN I, KANSAS CITY
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DURST JR, RDBERT D, TOPEKA
DUYSAK, SAMI, LEAVENWDRTH
DYCK, ARTHUR H, MCPHERSON
DYCK, CORA E, WICHITA
DYCK, ERIC LEE, HAYS
DYCK, GEORGE, NEWTON
DYER, VERNDN E, WICHITA
DYSART, JACK C, STERLING

E

EASTES, GARY DEAN, HALSTEAD
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EATON, GLEN E, SALINA
EATON, LESLIE F, SALINA
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EDROZO, M LUZ LUNA, COFFEYVILLE
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EDWARDS, MANIS C, WICHITA
EGEA, FERNANDO M, KANSAS CITY
EGELHOF, RICHARD H, WICHITA
EICHHDRN, FRANK D, GARDEN CITY
EIDT, DAVID W, OLATHE
EIDT, LAURENCE A, OLATHE
EIKERMANN, WILLIAM C, SHAWNEE MISSION
ELDER, DOUGLAS M, TDPEKA
ELDREDGE, LOUIS D, RUSSELL
ELLEDGE, E FRED, GREAT BEND
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ELLIS, HARVEY D, WICHITA
ELLIS, STEPHEN S, COFFEYVILLE
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EMPSON, CHARLES L, INDEPENDENCE
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ENNS, JAMES H, LAKE HAVASU CTY, AZ
ENOCH, ROLLAND, WICHITA
ENRIQUEZ JR, ROMAN S, KANSAS CITY
ENS, GERHARD GEORGE, HILLSBORO
ENS, PETER, HILLSBORO
ENSEY, T CRANFORD, MARION
EPLEE, JOHN P, WICHITA
ERICKSON, CLARENCE W, PITTSBURG
ERKEN, RONALD V, WICHITA
ESCH, JOHN G, PITTSBURG
ESRIG, HAROLD L, D.O., SHAWNEE MISSION
*ESTEP, THOMAS H, WICHITA
ESTRADA, EDMUNDO C, ANDOVER
ESTRADA, LINA, ANDOVER
ETZENHOUSER III, RUSSELL D, SHAWNEE MISSION
EVANS, FARRIS D, WICHITA
EVANS, GRANT E, WICHITA
*EVANS, JOHN F, WICHITA
EVANS, RICHARD W, MILWAUKEE, WI
EVANS, ROGER WILLIAMS, WICHITA
EVANS, WILLIAM R, GREAT BEND
EVANS JR, WILLIAM E, SHAWNEE MISSION
EWING, THOMAS D, LARNED
EYSTER, ROBERT L, WICHITA

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FABIAN, CAROL J, KANSAS CITY
FAIRCHILD, JDHN A, MANHATTAN
FAIRCHILD, RICHARD S, TOPEKA
FALLDN, JDHN H, KANSAS CITY, MD
FALTER, RICHARD T, HUTCHINSDN
FARHA, GEORGE J, WICHITA
FARHA, S JIM, WICHITA
FAST, RDBERT E, ATCHISDN
FAST, W SPENCER, ATCHISDN
FEAGAN, JERRY, TDPEKA
FELDMEYER, SEELEY T, SYRACUSE
FENDER JR, THOMAS H, WICHITA
FENT, LEE S, NEWTON
FENTON, ROBERT M, GARDEN CITY
FERGUSON, ROBERT LEDN, SHAWNEE MISSION
FERNANDEZ, HECTOR O, HDISINGTON
FERNANDEZ, LUIS A, TOPEKA
FERNIE, ROBERT W, BOULDER, CO
FERREE, RICHARD ALLAN, MCPHERSON
FERRELL, DDNALD P, WICHITA
FERRIS, BRUCE G, WICHITA
FESTOFF, BARRY W, KANSAS CITY, MD
FEUILLE JR, EDMDND G, WICHITA
FIELD, RICHARD A, TOPEKA
FIELDS, GALEN W, SCOTT CITY
*FIELDS DO, STEPHEN A, WICHITA
FIELDSTONE, PAUL R, JUNCTION CITY
FIESER, CARL W, GREAT BEND
FILLEY, VERNON W, PRATT
*FILLEY, WARREN, OKLAHOMA CITY, OK
FILLMAN, ELDDN M, TOPEKA
FINK, ABRAHAM A, PLANTATION, FL
FINLEY, DENNIS R, WICHITA
FISCHER, REX R, MANHATTAN
FISHER, JAMES B, WICHITA
FISHER, RAY F, WICHITA
FISHER, RONALD M, MANHATTAN
FITZGERALD, EDWARD J, WICHITA
FITZGERALD, THOMAS J, BAXTER SPRINGS
FITZGERALD MD, E JAMES, GODDLAND
FITZIG, SANFORD, WICHITA
FITZPATRICK, M ROBERT, KANSAS CITY
FLANDERS, H ALDEN, MISSION, TK
FLECKENSTEIN, CHARLES S, ONAGA
FLEMING, FORNEY W, WICHITA
FLESKE, LEONARD T, GREAT BEND
FLOERSCH, HUBERT M, KANSAS CITY
FLOWERS JR, CLELL B, WICHITA
FORD, CHARLES R, WICHITA
FORD, FRED L, TOPEKA
FORDYCE, NORMAN, SHAWNEE MISSION
FORET, JOHN D, KANSAS CITY
FORRED JR, WALTER A, HARPER
FORSTER JR, LDUIS G, SALINA
FORTUNE, CEDRIC B, OLATHE
FOSS, DANIEL C, HUTCHINSON
FOSTER, CHARLES G, TOPEKA
FOSTER, D BERNARD, TOPEKA
FOWLER, DENNIS L, WINFIELD
FOWLER, ROBERT J, WICHITA
FOWLER, WAYNE L, CONCORDIA
FOK, DEANNA K, KANSAS CITY
FOK, HOWARD A, KANSAS CITY
FRANCIS, NORTON L, WICHITA
FRANCISCO, CLARENCE L, SHAWNEE MISSION
*FRANCISCO, DAN A, WICHITA
FRANCISCO, EDGARDO, HORTON
*FRANCISCO, LINDA L, WICHITA
FRANCISCO, W DAVID, KANSAS CITY
FRANSEN, HERBERT, NEWTON
FRANSEN, PAUL H, NEWTON
FRANZ, ROBERT G, HILLSBORO
FRAZIER, RICHARD L, EMPORIA
FREDERICK, M F, HUGOTON
FREDRICKSON, DUANE E, LINDSBORG
FREEBORN JR, WARREN S, CLYDE
FREEMAN, F GILES, PRATT
FREEMAN, FRED A, MANHATTAN
FREEMAN, MALCOLM C, PITTSBURG
FREEMAN, RAYMOND S, SALINA
FRENCH, JAMES E, WICHITA
*FRENCH, JEROME E, WICHITA
FRIESEN, DALE, LAWRENCE
FRIESEN, FLDRENCE V, HESSTON
FRIESEN, ORLANDO J, BUHLER
FRIESEN, STANLEY R, KANSAS CITY
FRIGGERI, ROBERT W, GIRARD
FRITZ, GEORGE E, WICHITA
FRITZEMEIER, WILLIAM H, WICHITA
*FROMER, JOEL, WICHITA
FROMM, ARTHUR H, WICHITA
FRY, LUTHER L, GARDEN CITY
FULLEN, JERYL G, SHAWNEE MISSION
FULLER, DERYL D, LINDSBORG
FULTON, JOHN K, WICHITA
FUNK, EDWARD D, LINWOOD
FUSILLO, MICHAEL, TOPEKA

G

GALICHIA, JOSEPH P, WICHITA
GALLEHUGH, KEITH W, SHAWNEE MISSION
GALVAN, ALONSO, WICHITA
GANDHI, SHANTIKUMAR K, TOPEKA
GANN, E LAMONTE, EMPORIA
GANS, FREDERICK A, SALINA
GANZARAIN, RAMDN C, TDPEKA
GARCIA, FRANCISCO, SHAWNEE MISSION
GARCIA, GDULD C, EMPDRIA
GARCIA, GUILLERMD D, DODGE CITY
GARDINER, TED M, GARDEN CITY
GARDNER, BILLIE L, HARPER
GARDNER, GLENN M, SHAWNEE MISSIDN
GARDNER, JAMES D, MANHATTAN
GAREY, WILLIAM JDHN, INDEPENDENCE
GATEND, JDSEPH, GREAT BEND
GAY, JDHN D, TDPEKA
GEHRT, EARL B, CHANUTE
GEITZ, JAMES M, EMPDRIA
GELVIN, E RAYMOND, CONCORDIA
GENCH, RAYMOND L, CARMEL, CA
GENDEL, JOSEPH E, TOPEKA
GENILO, AMANCIO C, WICHITA
GENILO, CELESTE A, WICHITA
GENTRY, KALE C, SHAWNEE MISSION
GEORGE, EARL F, WICHITA
GEORGE, M DON, WICHITA
GERBER, ALLEN D, WICHITA
GERBER, HARRY A, LEAVENWORTH
GERJARUSAK, PRAPAS, KANSAS CITY
GESSLER, DONALD J, WICHITA
GETTLER, DEAN T, FORT SCOTT
GIAP, HAI PHUC, KNOKVILLE, IA
GIBBS, EUGENE, COFFEYVILLE
GIESSEL, MICHAEL D, TOPEKA
GILBERT, J HOWARD, SENECA
GILBERT, ROBERTA M, SHAWNEE MISSION
GILBERT II, JDHN H, GARDEN CITY
GILHOUSEN, FREDERIC M, KANSAS CITY
GILL, GEORGE L, LAMPE, MO
GILLEN, BILLY A, SHAWNEE MISSIDN
GILLES, HELEN M, LAWRENCE
GILMARTIN, RICHARD C, WICHITA
GIMPLE, KENNETH, TOPEKA
GINAVAN, DUANE A, EMPORIA
GIROD, CHARLES I, EL DORADO
GIRON JR, LOUIS T, KANSAS CITY
GIVNER, DAVID, WICHITA
GLAZZARD, CHARLES D, OLATHE
GLEASON, JIMMIE A, TOPEKA
GLENN, JAMES N, EMPORIA
GLENN, LYLE G, PROTECTION
GLOERSEN, PETER F, KANSAS CITY
GLOVER, RICHARD M, NEWTON
GNAU, FREDRIC B, HALSTEAD
GODFREY, KENNETH E, TOPEKA
GODFREY, ROBERT G, KANSAS CITY, MD
GODFREY, WILLIAM A, KANSAS CITY
GODWIN, PHILLIP A, LAWRENCE
GOERING, DONALD D, COLDWATER
GOERING, EMIL L, BURLINGAME
GOERTZ, KENNETH K, KANSAS CITY
GOERTZ, LEO R, KANSAS CITY
GOHIL, MAHENDRA N, WICHITA
GOLDBERG, HERBERT R, WICHITA
GOLDSTEIN, GERALD L, SHAWNEE MISSION
GOLLERKERI, MDHAN P, SHAWNEE MISSION
GOLLIER, ROBERT A, OTTAWA
GOLLIER II, ROBERT A, OTTAWA
GOMETZ, MODESTO S, PITTSBURG
GOMEZ, FRANCISCO, SHAWNEE MISSION
GONZALEZ, HIRAM, WICHITA
GOOD, JAMES T, FORT SCOTT
GOOD, MICHAEL W, CLAY CENTER
GOOD, WENDELL LISLE, SHAWNEE MISSION
GOODPASTURE, HEWITT C, WICHITA
GOODPASTURE, WILLARD C, HUTCHINSDN
GOODWIN, DONALD W, KANSAS CITY
*GOODWIN, MARY K, ANDALE
GOOTEE, JOSEPH E, TOPEKA
GOSALIA, ANIL V, SHAWNEE MISSION
GOTO, HIROSHI, KANSAS CITY
GOULDNER, RENE M, WICHITA
GOYLE, KRISHAN K, WICHITA
GOYLE, VIMAL, WICHITA
GRABER, CHARLES, NEWTON
GRADY, KENNETH L, KANSAS CITY
GRAHAM, KENNETH L, LEAVENWDRTH
GRAHAM, SUSAN B, SHAWNEE MISSIDN
GRAHAM, THOMAS W, LEAVENWORTH
GRAHAM JR, CHARLES P, TOPEKA
GRANTHAM, JARED J, KANSAS CITY
GRAUEL, CHARLES W, WICHITA
GRAVES, JACK W, WICHITA
GRAVES, KATHRYN, HUTCHINSON
GRAY, C LUCIEN, WICHITA
GRAY, DAVID E, TOPEKA
GRAY, H TOM, WICHITA
GRAYIB, ANTOINE S, TOPEKA
GRAYSON, ROY D, ALBUQUERQUE, NM
GREEN, LAWRENCE C, ARKANSAS CITY
GREENBERG, GEORGE E, DODGE CITY
GREENBERG, MARK, TOPEKA
GREENBERGER, N J, KANSAS CITY
GREENE, HORACE T, TOPEKA
GREENWOOD, EDWARD D, TOPEKA
GREENWOOD, JAMES F, GARDEN CITY
GREER, JAMES A, WICHITA
GREER, RICHARD H, TOPEKA
GRIBBLE, ROBERT N, WICHITA
GRIFFITH, FRANK H, SALINA
GRILLOT, FLOYD B, WICHITA
GRIMALDI, GARY A, FORT SCOTT
GRIMES, I ROSS, LIBERAL
GRIMES, JAMES T, LYONS
GRISOLIA, ANDRES, LEAVENWORTH

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GRISWOLD, DALE G, NEWTON
GROHS, HEINZ K, WICHITA
GROSSMAN, HARVEY M, SHAWNEE MISSION
GROVE, JOHN A, NEWTON
GROWNEY, JOHN T, ATCHISON
GRUENDEL, RICHARD A, KANSAS CITY
GRUENDEL, VIRGINIA T, KANSAS CITY
GRUND, FRANK M, WICHITA
GRUNDMEIER, ANNETTE M, SHAWNEE MISSION
GRUSHNYS, ARNOLD, WICHITA
GSELL, GEORGE F, WICHITA
GUARDIA, DAVID K, KANSAS CITY
GUERRA, TOMAS H, MARION
GUNN, MARVIN R, SALINA
GUNTER, CARL C, QUINTER
GUTHRIE, RICHARD A, WICHITA
GUTOVITZ, ALLEN LOUIS, TOPEKA
GUTTIKONDA, PRASAD B, WICHITA
GUZMAN, MANUEL, SALINA

H

HA, SANG W, COFFEYVILLE
HABASHY, SHAWKY N F, WICHITA
HACKER, DAVID CHARLES, SHAWNEE MISSION
HACKER, ELAINE MARY, TOPEKA
HADLEY, DELMONT C, OTTAWA
HAFFNER, WILLIAM N, EL DORADO
HAGAN, C THOMAS, WICHITA
HAGAN, FRANCIS J, WICHITA
HAGGAN, MARGARET E, LAWRENCE
HAIGLER, JAMES P, HAYS
HAINES, ROBERT A, TOPEKA
HAIRE, WILLIAM O, HALSTEAD
HALE, RALPH, WICHITA
HALL, J ROGER, WICHITA
HALL, JERRY O, ANTHONY
HALL, WESLEY H, GIRARD
HALL III, THOMAS BRYAN, KANSAS CITY
HALL JR, ERNEST B, HIAWATHA
HALLEY, M MARTIN, TOPEKA
HALLING, L WILLIAM, HAYS
HALPIN, EDWARD D, WICHITA
HALVORSON, HOWARD C, OLATHE
HAMILTON, JAMES J, WAKEENEY
HAMM, ORVAL L, PAKISTAN,
HAMMEKE, JOHN C, LEAVENWORTH
HAMMEL, GEORGE W, BELLA VISTA,AR
HAMTIL, LAWRENCE W, SHAWNEE MISSION
HAN, CHAN S, COFFEYVILLE
HANCOCK, ALAN C, KANSAS CITY
HANCOCK, DANIEL E, MANHATTAN
HANDLEY, DENNIS MICHAEL, SHAWNEE MISSION
HANSA, HANSA, HAYS
HANSON, DAVID C, IOLA
HANSON, FRANK W, GARDEN CITY
HANSON, FREDERICK, KANSAS CITY
HAQUE, ANWAR U L, KANSAS CITY,MO
HARA, GLENN S, KANSAS CITY
HARBIN, GARY LYNN, SALINA
HARD, BENJAMIN F, KANSAS CITY,MO
HARDIN, CREIGHTON A, KANSAS CITY
HARMS, ALBERT C, SHAWNEE MISSION
HARMS, EDWIN M, WICHITA
HARMS, WILMER A, HALSTEAD
HARRIS, FRANK H, WICHITA
HARRIS, HUBERT L, TOPEKA
HARRIS, JO ANN SPEIGEL, KANSAS CITY
HARRIS, NORMAN R, SALINA
HARRIS, NORVAN O, LIBERAL
HARRIS, PATRICIA A, TOPEKA
HARRIS JR, CLAIB B, GARNETT
HARRISON, A BROOKS, WICHITA
HARRISON, HALL E, TOPEKA
HARRISON, PAUL BARRY, WICHITA
HART, DILLIS L, WICHITA
HART, KELLY Z, KANSAS CITY
HART, LAWRENCE E, ATCHISON
HART JR, PAUL V, KANSAS CITY
HARTLEY, FOUNT K, GAINESVILLE,FL
HARTLEY, JAMES M, WICHITA
HARTLEY, ROY WILEY, NORTON
HARTMAN, CHARLES R, KANSAS CITY
HARTMAN, GERALD V, KANSAS CITY
HARTMAN, ROGER L, NORTON
HARTONG, WILLIAM A, SHAWNEE MISSION
HARVEY, JOHN E, EMPORIA
HARVEY, ROSEMARY B, WICHITA
HARWOOD, CLAUDE J, GLASCO
HASKINS, ROBERT J, CHANUTE
HASSAN, RIZWAN U, WICHITA
HASSELLE III, JAMES E, LAWRENCE
HASSETT, GERARD R, COLBY
HASSLER, RANDY O, SALINA
HASTINGS, MARY T, SHAWNEE MISSION
HATHAWAY, PETER, SHAWNEE MISSION
HATTON, DONALD W, LAWRENCE
HATTON, LLOYD W, SALINA
HATTRUP, RICHARD J, WICHITA
HAVENHILL II, MARSHALL A, EMPORIA
HAWLEY, RAYMOND G, WICHITA
HAYES, KRIS A, WICHITA
HAYES, WILLIAM L, WICHITA
HAYS, THOMAS H, WICHITA
HEASTY, ROBERT G, MANHATTAN
HEBBAR, SATYA N, TOPEKA
HEDRICK, KENNETH E, HUTCHINSON
HEIDGEN, DEBRA L, SHAWNEE MISSION
HENDERSON, CHARLES F, PARSONS
HENDERSON, DAVID V, GARNETT
HENDRICKSON, JON R, NEWTON
HENDRICKSON, KATHRYN O, NEWTON
HENNING, CALVIN W, OTTAWA
HENNING, CHARLES E, WICHITA
HENNING JR, HAROLD JOHN, KANSAS CITY
HENRY, JOSEPH E, SHAWNEE MISSION
HENSLEY JR, CLINE O, WICHITA
HEREDO, JOHN, WICHITA
HERMES, RICHARD L, LAWRENCE
HERMRECK, ARLO S, KANSAS CITY
HERNANDEZ, MANUEL, ARKANSAS CITY
HERRERA, JORGE J, TOPEKA
*HERSHBERGER DO, GROVER, WICHITA
HERSHORN, SIMON E, WICHITA
HESSE, FREDERICK J, SALINA
HESSER, HERBERT H, SHAWNEE MISSION
HETTINGER, MICHAEL E, KANSAS CITY
HIEBERT, ABRAHAM E, WICHITA
HIEBERT, DAVID L, LAWRENCE
HIEBERT, JOHN B, TOPEKA
HIEBERT, JOHN M, KANSAS CITY
HIEBERT, PETER E, SHAWNEE MISSION
HIESTERMAN, HERMAN W, QUINTER
HILOYARD II, VICTOR H, COLBY
HILL, JAMES E, BELLA VISTA,AR
HILL, JOHN J, COLORADO SPRING,CO
HILL, LARY MICHAEL, GREAT BEND
HILL, RICHARD H, MEADE
HILL, ROBERT N, TOPEKA
HILL, ROONEY W, SHAWNEE MISSION
*HILST, WILBUR O, TOPEKA
HINSHAW, ALFRED H, WICHITA
HINSHAW, CHARLES T, WICHITA
HINSHAW, EDGAR O, ARKANSAS CITY
*HINSHAW JR, CHARLES T, WICHITA
HINTHORN, DANIEL R, KANSAS CITY
HIRATZKA, TOMIHARU, WICHITA
HIRD, WAYNE E, LAWRENCE
HIRSCHBERG, J COTTER, TOPEKA
HISZCZYNSKYJ, ROMAN, TOPEKA
HITCHCOCK, C THOMAS, KANSAS CITY
HIZON, RAMON R, WICHITA
HOADLEY, WILLIAM O, KANSAS CITY
HOBBS, DONALD O, TOPEKA
HOBSON, MILBURN W, SHAWNEE MISSION
HODES, HERBERT C, SHAWNEE MISSION
HODGES, BRUCE E, SHAWNEE MISSION
HODGES, GLENN R, KANSAS CITY
HODGES, MERLE A, SALINA
HODGSON, DAVID K, WASHINGTON
HODGSON, ROBERT B, WICHITA
HODSON, HERVEY R, WICHITA
HOFER, DEWAYNE O, CONCORDIA
HOFFER, JOHN G, MEDICINE LODGE
HOFFMAN, CARL, COFFEYVILLE
HOHERZ, DAVID G, TOPEKA
HOHLY, EVE K, KANSAS CITY,MO
HOLCOMB, DONALD G, LOS ANGELES,CA
HOLCOMB, WILLIAM M, LIBERAL
HOLOCRAFT, JACQUELYNE, KANSAS CITY
HOLDEN, DAVID M, WICHITA
HOLDEN JR, RAYMOND F, WICHITA
HOLDERMAN, WALLACE O, HUTCHINSON
HOLLADAY, HARMON M, WICHITA
HOLLADAY, KENNETH R, EUDORA
HOLMAN, JON B, SALINA
HOLMES, FREDERICK F, KANSAS CITY
HOLMES, GRACE E, KANSAS CITY
*HOLMES, JED, WICHITA
HOLMES, JOHN A, KANSAS CITY
HOLMES, ROBERT W, TOPEKA
HOLSINGER, DONALD M, PITTSBURG
HOLT, JOHN M, GREAT BEND
HOMPLUEM, PACHAREE, PARSONS
HOOFER, WILFORD O, HALSTEAD
HOOVER, CYNTHIA J, KANSAS CITY
HOPKINS, WILLIAM O, SHAWNEE MISSION
HOPKINS JR, B MORRISON, SCOTT CITY
HOPPER, CHARLES R, EMPORIA
HORBELT, DOUGLAS V, WICHITA
HORNE, JAMES B, TOPEKA
HORSEMAN, ROBERT F, SHAWNEE MISSION
HORTON, BILL GATE, LAWRENCE
HORTON, WILLIAM A, KANSAS CITY
HOSTETTER, JAMES P, TOPEKA
HOSTETTER, PHILIP H, MANHATTAN
HOUSHOLDER, DANIEL FAIR, WICHITA
HOUSHOLDER, MARTHA S, WICHITA
HOUSTON II, LAWRENCE MORLEY, SHAWNEE MISSION
HOWARD, DONALD O, WICHITA
HOWELL, BARBARA JOYCE, EMPORIA
HOWERTER JR, BERNARD E, COFFEYVILLE
HOYLE, WINTHROP E, ESTES PARK,CO
HOYT, ARTHUR W, TOPEKA
HSU, CHENG H, TOPEKA
HSU, SHIN-FU, TOPEKA
HUAMAN, ANTONIO M, *,
HUANG, GEORGIANA L W, SHAWNEE MISSION
HUANG, JONSON, TOPEKA
HUDSON, JAMES R, EDMOND,OK
HUDSON, ROBERT P, OLATHE
HUEBERT, DEAN A, WICHITA
HUEBNER, ROBERT STEPHAN, PITTSBURG
HUERTER, DAVID F, PITTSBURG
HUERTER, QUENTIN C, KANSAS CITY
HUGHES, ROBERT WALTER, LAWRENCE
HULL, KENNETH L, WICHITA
HULTGREN, MYRON K, WICHITA
HUME, JOSEPH W, WICHITA
HUMMER, LLOYD M, WICHITA
HUMPHREY, LOREN JENKINS, SHAWNEE MISSION
*HUNO, LARRY R, WICHITA
HUNTER, KENNETH R, LEBO
HUNTER JR, JAMES S, MANHATTAN
HURWITZ, ARYEH, KANSAS CITY
HUSEMAN, RICHARD ALLAN, NORTH KANS CITY,MO
HUSTEAD, ROBERT F, WICHITA
HUSTON, FRANCIS W, WINCHESTER
HUSTON, JOSEPH W, TOPEKA
HUTCHINSON, DIRK T, WINFIELD
HUTCHISON, GLEN C, HAYS
HUTTON, FREDERICK A, TOPEKA
HWA, EUGENE C, NEWTON
HYLAND, JOSEPH M, TOPEKA
HYNES, HARRY E, WICHITA

I

IBARRA, J LUIS, WICHITA
IBARRA, RICHARD C, KANSAS CITY
IOBEIS, BAOR, WICHITA
IHRIG, ROGER W, HUGOTON
ILIFF, R DOUGLAS, TOPEKA
*ILORETA, ALFREDO T, TOPEKA
INGHAM JR, H LAIRD, LAWRENCE
INGRAM, JOHN E, KANSAS CITY
INNES, ROBERT C, KANSAS CITY
IOANNOU, NICHOLOAS, WICHITA
IRBY, ADDISON C, FORT SCOTT
IRBY, PRATT, FORT SCOTT
IRWIN, RICHARD L, NEWTON
ISAAC, CHARLES A, NEWTON
ISAACSON, RICHARD N, TOPEKA
ITURRALDE, GEORGE, SHAWNEE MISSION
IWAY, BELINDA D, ELKHART
IWAY, OLIVIA N, ELKHART

J

JABEL, JUVENAL T, SATANTA
JACKMAN, GLENN H, ESTES PARK,CO
JACKS, J WARREN, HAWASEE,AZ
*JACKSON, CATHERINE M, WICHITA
JACKSON, CHARLES R, WICHITA
JACKSON, LINDA H, TOPEKA
JACKSON, ROBERT V, SHAWNEE MISSION
JACKSON, ROGER PAUL, N KANSAS CITY,MO
JACKSON, VICTOR L, ALTAMONT
JACKSON JR, DELMAS A, SALINA
JACOB, KANNAMPALLY L, EL DORADO
JACOBS, DAVID S, KANSAS CITY
JACOBS, RAE R, KANSAS CITY
JACOBSEN, DWIGHT SKINNER, COLBY
JACOBSON, JOEL A, WAKEENEY
JACOBY II, ROBERT E, TOPEKA
JAHANIAN, DARYOUSH, KANSAS CITY
*JAMES, DONALD L, WICHITA
JAMES, VERNON L, WICHITA
JANES, DONALD R, SHAWNEE MISSION
JANSSEN, ERWIN T, TOPEKA
JANZEN, HERMAN F, MOUNDRIDGE
JARROTT, JOHN B, HUTCHINSON
*JAWADI, JAMEELA HUSAIN, WICHITA
JAYARAM, MARANDAPALLI R, KANSAS CITY
JAZAYERLI, NABIL, WICHITA
JEHAN, SAYED S, WICHITA
JENNEY, CHARLES B, WICHITA
JENSEN, THOMAS M, OLATHE
JESTER, SHELBY L, WICHITA
JEWELL, ROSS L, ST FRANCIS
JEWELL, WILLIAM R, KANSAS CITY
JIRICKO, MILOS, COFFEYVILLE
JOHLER, TERRY HARTWIG, LEAVENWORTH
JOHNS JR, LEO E, KANSAS CITY,MO
JOHNSON, BRUCE E, KANSAS CITY
JOHNSON, CAROL ANN, WICHITA
JOHNSON, CYNDA A, KANSAS CITY
JOHNSON, EDEN E, TOPEKA
JOHNSON, FREDERICK E, KANSAS CITY,MO
JOHNSON, GEORGE K, WICHITA
JOHNSON, HOWELL O, OOOGE CITY
JOHNSON, J RICHARD, MCPHERSON
JOHNSON, JOHN E, KANSAS CITY
JOHNSON, NADINE KAY, SHAWNEE MISSION
JOHNSON, PAUL O, LEAVENWORTH
JOHNSON, RANDLE C, HUTCHINSON
JOHNSON, THOMAS E, WICHITA
JONES, CHARLES E, SHAWNEE MISSION
JONES, EDWARD L, GREAT BEND
JONES, FORREST H, COLUMBUS
JONES, H IVOR, SHAWNEE MISSION
JONES, H PENFIELD, LAWRENCE
JONES, JAY S, WICHITA
JONES JR, HERMAN H, KANSAS CITY
JOSEPH, BRIAN W, TOPEKA
JOSEPH, HOWARD F, LAWRENCE
JOSS, CHARLES S, TOPEKA
JOUVENAT, NEIL C, SHAWNEE MISSION
JOYCE, G BERNARD, TOPEKA
JUBELT, HILBERT P, MANHATTAN
JUDILLA JR, FRANCISCO, WICHITA
JUSTUS, WILLIAM J, PLEASANTON

* Probationary members.

K

KADIAN, RAJESH S, SHAWNEE MISSION
KADISON, HERBERT I, WICHITA
KAGAN, STUART M, SHAWNEE MISSION
KALBAC, RICHARD W, GARDEN CITY
KALOOR, RICHARD H, MANHATTAN
KALIVAS, JAMES T, KANSAS CITY
KANE JR, WILLIAM M, HAYS
KARDATZKE, E STANLEY, WICHITA
KARDATZKE, JON K, WICHITA
KARLIN, CHARLES A, SHAWNEE MISSION
KARLIN, RONALD J, SHAWNEE MISSION
KARNAZE, MICHAEL G, KANSAS CITY
KASHA, ROBERT L, WICHITA
KASHYAP, BANSHI PRASAD, SHAWNEE MISSION
KASSEBAUM, GLEN E, EL DORADO
KASSEBAUM, KENNETH G, WICHITA
KATZ, JEROME B, TOPEKA
KAUFMAN, EUGENE E, WICHITA
KAUFMAN, LELAND R, WINFIELD
KAUFMAN, WILLARD E, MOUNDRIDGE
KAUL, ANAND N, WINFIELD
KAVEL, KARL K, TOPEKA
KAVI, NAGESH G, LEAVENWORTH
KEENE, GEORGE H, WICHITA
KEIL, JAMES E, LARNED
KEITH, ROBERT MARSHALL, EMPORIA
KELLER, JAMES P, WICHITA
*KELLERMAN, RICK, WICHITA
KELLING, COLLYER, EMPORIA
KELLY, DAN A, TOPEKA
KELLY, ROBERT W, WICHITA
KEMPTHORNE, CHARLES R, MANHATTAN
KENDALL, TOM E, WICHITA
KENDRICK, J GILLERAN, WICHITA
KENNALLY, KEVIN P, SABETHA
KENNEDY, GERALD T, WICHITA
KENNEDY, HOWARD U, TOPEKA
KENNEDY, JAMES A, KANSAS CITY
KENNEDY, KENNETH R, SHAWNEE MISSION
KEPES, JOHN J, KANSAS CITY
KEPKA, DENNIS, HUTCHINSON
KERBY, GERALD R, KANSAS CITY
KESTENBAUM, THELDA M, KANSAS CITY
KETCHERSIDE JR, WILLIAM J, KANSAS CITY
KETCHUM, LYNN O, SHAWNEE MISSION
KEYS JR, ROBERT C, TOPEKA
KEYS SR, ROBERT C, TOPEKA
KHICHA, GYANCHAND J, WICHITA
KHOURY, GEORGE H, WICHITA
KHURANA, SATISH K, SHAWNEE MISSION
KIFER, C JAMES, HAYS
KIHM, ALBERT A, CHANUTE
KIM, JONG M, KANSAS CITY
KIM, PAIK N, WICHITA
KIM, YONG W, TOPEKA
KIMBALL, RICHARD R, MANKATO
KIMBLE, JAMES A, WICHITA
KIMURA, CHARLES C, SHAWNEE MISSION
KINDLING, PAUL H, TOPEKA
KING, CHARLES R, KANSAS CITY
KING, WILLIAM T, GREAT BEND
KINNAN, L F, CALDWELL
KINPORTS JR, EDWARD D, KANSAS CITY,MO
KINPORTS SR, EDWARD B, KANSAS CITY,MO
KIRBY, MERLIN G, GREAT BEND
KIRCHNER, FERNANDO R, KANSAS CITY
KIRK, THOMAS E, MANHATTAN
KIRK JR, E DAVID, WICHITA
KIRKEGAARD, RODGER S, TOPEKA
KISER, JOHN L, WICHITA
KISER, WILLARD J, WICHITA
KISHORE, ROY N, PARSONS
KISHORE, SHEELA, PARSONS
KITCHEN, ROBERT R, WICHITA
KLEINHOLZ JR, EMIL JOHN, TOPEKA
KLEMMER, HERBERT, TOPEKA
KLENDA JR, MARTIN B, BELOIT
KLIEWER, VERNON L, NEWTON
KLINGLER JR, EUGENE A, MANHATTAN
KLOBASA, CHARLES L, MANHATTAN
KLONTZ, WILLIAM JOSEPH, WICHITA
KLOSTERHOFF, BRUCE E, HUTCHINSON
KNAPP, LESLIE E, WICHITA
KNAPP, M ROBERT, WICHITA
KNAPPENBERGER, ROY C, COLORADO SPRING,CO
KNECHT, STEPHEN M, EMPORIA
KNEIDEL, THOMAS W, WICHITA
*KNIGHT, PHILIP J, WICHITA
KNUTH, KENNETH L, INDEPENDENCE
KODANAZ, A AYTEKIN, SHAWNEE MISSION
KOKSAL, TOM, GARDEN CITY
KOONS, JESS W, LIBERAL
KOSAR, CLARENCE D, CONCORDIA
KOURI, SAMMY H, WICHITA
KOVARIK, ERNEST O, TOPEKA
KOZIKOWSKI, BEN M, SHAWNEE MISSION
KRANTZ, KERMIT E, KANSAS CITY
KRAUSE, ROLAND L, WICHITA
KREHBIEL, MARK A, SALINA
KRETSINGER OD, W BROCK, EMPORIA
KROLL, HARRY G, TOPEKA
KRUCKEMYER, ALAN L, SALINA
KRUEGER, HAVEN C, GREAT BEND
KRUEGER, KURT ALLEN, SHAWNEE MISSION
KRUMME, BARBARA J, KANSAS CITY
KRUPKA, JOHN J, WICHITA
KUBIN, DORIS A, SHAWNEE MISSION
KUBINA, GLENN RICHARD, WICHITA
KUMAR, SURINDER, NEWTON
KURTH, C JOSEPH, WICHITA
KURTH, PAUL H, SHAWNEE MISSION
KURTH, ROBERT H, SHAWNEE MISSION
KUTILEK, FRANK J, WICHITA
KWEE, SIOE T, KANSAS CITY
KYNER, JOSEPH L, KANSAS CITY

L

LABHSETWAR, S A, JUNCTION CITY
*LACCHEO, MICHAEL L, TOPEKA
*LACEY, STEFFAN R, WICHITA
LAFENE, BENJAMIN W, MANHATTAN
LAHAM, ALEXANDER J, DALLAS,TX
LAI, CHI-WAN, KANSAS CITY
LAI, JENG Y, WICHITA
*LAI-CHEN, SHEUE-HUEY, TOPEKA
LAING, ROBERT R, KANSAS CITY
LAIRD, DALE D, OLATHE
LAKE, MAX S, SALINA
LANCE, RAYMOND W, PITTSBURG
LANCE JR, JOHN F, WICHITA
LANSKY, LESTER L, KANSAS CITY
LANSKY, SHIRLEY B, KANSAS CITY
LAPI, RUTH M, SHAWNEE MISSION
LARSON, DELBERT L, HIAWATHA
LASLEY, DAVID A, SALINA
LASLEY, MICHAEL B, HAYS
LATIMER, KATHERINE, WICHITA
LAUNEY, WALTON S, TOPEKA
LAURY, DAVID G, OTTAWA
LAUVER, MARY ANN, WICHITA
LAVA, CHIRUNO, PARSONS
LAVIN, MARK J, KANSAS CITY
LAW, FINDLEY, ELLINWOOD
LAWLESS, HAROLD L, BLUE RAPIDS
LAWN, CLAUDIA A, WICHITA
LAWN, RAYMOND A, WICHITA
LAWRY, JAMES VORIS, WINFIELD
LAWS, LEWIS R, MARYSVILLE
LAWSON, DWIGHT, TOPEKA
LAWTON, MARVIN K, CONCORDIA
LAWWILL, THEODORE, KANSAS CITY
LAYBOURNE JR, PAUL C, KANSAS CITY
LAZAR, HARRY, WICHITA
LEARNED, GEORGE R, LAWRENCE
LEATHERS III, HOLLIS K, SHAWNEE MISSION
LEE, CATHERINE, TOPEKA
LEE, JAE M, KANSAS CITY
LEE, KYO R, KANSAS CITY
LEE, R REX, WICHITA
LEE, SANG DUG, FT DODGE,IA
LEE, SONG DOW, TOPEKA
LEE, SONG PING, TOPEKA
LEE, YONG U, EL DORADO
LEE JR, EDWARD S, WICHITA
LEE JR, JAMES G, KANSAS CITY
LEFFLER, PAUL B, PITTSBURG
LEGASPI JR, PEDRO L, SHAWNEE MISSION
LEGER, LEE H, FT MYERS,FL
LEGLER DO, GARY LEE, TOPEKA
LEIFER, WILLIAM N, TOPEKA
LEIGH, LAWRENCE E, SHAWNEE MISSION
LEIKER, JOSEPH, ARKANSAS CITY
LEISY, JERALD W, WICHITA
LEITCH, DAVID A, GARNETT
LEMOINE JR, ALBERT N, KANSAS CITY
LENEVE, ROBERT T, HUGOTON
LENSKI JR, FRANCIS X, IOLA
LENTZ, WILLIAM R, TOPEKA
LEO, WILLIAM A, KANSAS CITY
LERNER, SCOTT A, SHAWNEE MISSION
LESSENDEN, GLENN A, LAWRENCE
LESSENDEN JR, C M, TOPEKA
LESSER, DANE A, HUTCHINSON
LESTER, JOHN BUCKLES, SHAWNEE MISSION
LETTNER, HANS T, HUTCHINSON
LEVINE, ERROL, KANSAS CITY
LEVINE, WILLIAM R, WICHITA
LEVY, EDWIN Z, TOPEKA
LEWIN, WALTER, SHAWNEE MISSION
LEWIS, JAMES E, SHAWNEE MISSION
LEWIS, LARRY O, WICHITA
LEWIS JR, H DANIEL, KANSAS CITY,MO
LIEBERMAN, BRUCE IRWIN, KANSAS CITY
LIES, BARTHEL N, COLWICH
LIES, RICHARD B, WICHITA
*LIESMANN, GEORGE E, TOPEKA
LIESMANN, JEAN ELIZABETH, TOPEKA
LIM, CARLO, SEDAN
LIN, JAMES T Y, KANSAS CITY
LIN, JOE J, WICHITA
*LINO II, EDWARD J, GARDEN PLAIN
LINDHOLM, GERALD R, NEWTON
LINDSLEY, CAROL B, KANSAS CITY
LINDSLEY, HERBERT B, KANSAS CITY
LINHAROT, RONALD D, WICHITA
LINN, CATHERINE P, KANSAS CITY
LINSHAW, MICHAEL A, KANSAS CITY
LIPOFF, JAY I, LEAVENWORTH
LIPSEY, JAMES H, SHAWNEE MISSION
LITTELL, JAMES A, WICHITA
LITTLE, L GILBERT, WICHITA
LIU, CHIEN, KANSAS CITY
LIVINGSTON, CHARLES E, SALINA
LLOYD, HARVEY L, KANSAS CITY
LLOYD, JAMES W, CONCORDIA
LOCKHART, JOSEPH G, WICHITA
LOEFFLER, JAMES A, WICHITA
LOEWEN, HENRY H, WICHITA
LOEWEN, PETER S, HILLSBORO
LOEWEN, WILLIAM C, WICHITA
LOGAN, GEOFFREY G, WICHITA
LOGANBILL, VAROEN J, MOUNDRIDGE
LOHMEYER, KENNETH L, EMPORIA
LONEY, JOHN M, BELOIT
LONG, EDWARD E, HUMBOLDT
LONG, JOHN W, TOPEKA
LONG, LLOYD O, GOODLAND
LONG, ROBERT C, NORTON
LOREN, MICHAEL LEWIS, SHAWNEE MISSION
*LOSEE, JOHN M, WICHITA
LOTITO, CARLOS A, KANSAS CITY
LOVELAND, G CHARLES, LAWRENCE
LOVETT, PAUL A, WICHITA
LOW, HAROLD L, WICHITA
LOWE, STANLEY W, MANHATTAN
LOWMAN, JAMES T, KANSAS CITY
LUEDTKE, WALTER E, EMPORIA
LUEGER OD, JAMES JOHN, SENECA
LUEKEN, LUEKE B, WICHITA
LUELLEN, THOMAS J, WICHITA
LUETJE, CHARLES MARION, KANSAS CITY,MO
LUKENS, DAVID, HUTCHINSON
LUKERT, BARBARA P, KANSAS CITY
LULO, ANTONIO R, SHAWNEE MISSION
LUMB, RAYMOND C, TOPEKA
LUNGSTRUM, JACK E, SALINA
LUZZATI, ENZO F, WICHITA
LYNCH, JOHN A, TOPEKA
*LYNCH, MARY A, WICHITA
LYNCH, SEAN R, KANSAS CITY
LYONS JR, FRANK C, MANHATTAN

M

MAC KILLOP JR, DANIEL, WINFIELD
MACARTHUR, RICHARD IAN, KANSAS CITY
MACE, RONALD D, JUNCTION CITY
MACY, NORMAN E, SALINA
MACY, TED L, SALINA
MADISON, WILLARD A, NORTONVILLE
MADISON JR, WARD N, WICHITA
MADSEN, GLENN L, LAWRENCE
MAGIDSON, ELLIOT ARTHUR, WICHITA
MAGRINA, JAVIER E, KANSAS CITY
MAILMAN, GERSHOM, WICHITA
MALIK, MUMTAZ ILAHI, WICHITA
MALLORY, JOHN A, SHAWNEE MISSION
MALONE, EUGENE M, HALSTEAD
MANAHAN, G EUGENE, LAWRENCE
MANGOLD, JOEL VOYCE, KANSAS CITY
MANI, MANI M, KANSAS CITY
MANLEY, JOSEPH W, SHAWNEE MISSION
MANNING, ROBERT T, WICHITA
MANSFIELD, CARL M, KANSAS CITY
MANSOUR, BADIE S, WICHITA
MANTZ, FRANK A, SHAWNEE MISSION
MARCHBANKS, DONALD L, SALINA
MARPLES, BRADLEY W, KANSAS CITY
MARR, GARRETT L, SHAWNEE MISSION
MARSH, ALICE GARRISON, KANSAS CITY
MARSH, CONNIE M, HALSTEAD
MARSH, HENRY D, WICHITA
MARSHALL, B M, TOPEKA
MARSHALL, GEORGE D, COLBY
MARSHALL, GEORGE W, SALINA
MARSHALL, RONALD L, MANHATTAN
MARTIN, DANIEL C, MANHATTAN
MARTIN, EARL A, PARSONS
MARTIN, JEFFERY L, MARION
MARTIN, JOSEPH P, KANSAS CITY
MARTIN, NORMAN L, KANSAS CITY
MARTIN, OLIVER L, SALINA
MARTIN, RONALD C, POTOMAC,MD
MARTIN, WILLIAM D, TOPEKA
MARTIN JR, GLEN E, WICHITA
MARTINEZ, JOHN D, KANSAS CITY
MARVIN, NORMAN G, SHAWNEE MISSION
MARYMONT JR, JESSE H, WICHITA
MASER, GEORGE R, SHAWNEE MISSION
*MASSEY, ANDREW D, WICHITA
MASTERS, FRANCIS W, KANSAS CITY
MASTERSON, MELVIN LEROY, TROY
MASTIO JR, GEORGE J, WICHITA
MATASSARIN, BENJAMIN M, WICHITA
MATASSARIN, FREDERICK W, WICHITA
MATHEWS, DAVID R, SHAWNEE MISSION
MATHEWS, ROBERT C, OLATHE
MATHEWS, ROBERT MAJOR, SHAWNEE MISSION
MATHEWSON, HUGH S, KANSAS CITY
MATHIS, JERRY L, SALINA
MATTHEW, WILLIAM L, OLATHE
MATTICK, IRVIN H, HAYS
MATTIOLI, LEONE, KANSAS CITY
MAU, WALTER, TOPEKA
MAUCK, HAROLD C, STOCKTON
MAWDSLEY, MICHAEL W, WICHITA
MAXFIELD, RUSSELL J, GARDEN CITY
MAXWELL, GORDON E, SALINA
MAXWELL, ROBERT A, SHAWNEE MISSION
MAY, KENNETH L, BONNER SPRINGS
MC GEENEY, TERRY L, SENECA
MC NICKLE, GEORGE ANDREW, WICHITA
MC PHEE, MARK S, KANSAS CITY
MCALLASTER, WENDALE E, GREAT BEND

* Probationary members.

*MCBRIDE, ANN, WICHITA
MCCANN, PATRICK E, FORT SCOTT
MCCANN, WILLIAM E, OLATHE
MCCARTER, DUANE K, TOPEKA
MCCARTHY, ROBERT P, KANSAS CITY
MCCAUGHEY, HUGH W, SHAWNEE MISSION
MCCLANAHAN, WARD A, WICHITA
MCCLELLAN, ERNEST L, WICHITA
MCCLELLAN, JOHN W, TOPEKA
MCCLURE, JAMES A, TOPEKA
MCCOLLUM, WILLIAM B, LEAVENWORTH
MCCOMAS JR, MARMADUKE O, TOPEKA
MCCOWEN, HERBERT M, KANSAS CITY
MCCOY, CHARLES P, WICHITA
MCCOY, CHARLES T, HUTCHINSON
MCCOY, MICHAEL T, TOPEKA
MCCOY, RONALD, DODGE CITY
MCCRAE, SPENCER C, SALINA
MCCULLOUGH, ROBERT C, GOODLAND
MCCUNE, MARK A, KANSAS CITY
MCEACHEN, WILLIAM H, SHAWNEE MISSION
MCELHINNEY, CHARLES F, DODGE CITY
MCELROY, MICHAEL B, SHAWNEE MISSION
MCELROY, ROBERT T, TOPEKA
MCELROY, WILBUR J, TOPEKA
MCGINNIS, MICHAEL J, DODGE CITY
MCGUIRE, WILLIAM F, WICHITA
MCGURK, THOMAS E, SHAWNEE MISSION
MCKEE, LEO F, COTTONWOOD FALS
MCKEE, RICHARD S, LEAVENWORTH
MCKENNA, MICHAEL J, FORT SCOTT
MCKERRACHER, ROBERT O, DERBY
MCKIM, W LYNN, KINSLEY
MCKNIGHT, DAVID E, MANHATTAN
MCKNIGHT, ELLIS B, ALMA
MCLAIN, KENNETH, RANSOM
*MCMULLEN, BRUCE R, WICHITA
MCMULLEN, JOSEPH E, HUTCHINSON
MCNEIL, ELBERT O, MANHATTAN
*MCQUEEN, DAVID ARNOLD, WICHITA
MCREYNOLDS, CHARLES R, WICHITA
MCVAY, P BRUCE, CLAY CENTER
MCWHERTER, LOTTIE B, SHAWNEE MISSION
MEADOWS, DONALD C, WICHITA
MEBUST, WINSTON K, KANSAS CITY
MECH, ARNOLD W, TOPEKA
MEDHAT, MOHAMED A, KANSAS CITY
MEDINA, ANIBAL, HAYS
MEE, ADRIAN W, OLATHE
MEEK, PALMER F, MANHATTAN
MEEK JR, JOSEPH C, KANSAS CITY
MEEKER II, BRUCE P, WICHITA
MEGIBOW, ALAN D, TOPEKA
MEHAFFY, ORVILLE A, BAXTER SPRINGS
MEIDINGER, RAY, HIAWATHA
MEIDINGER, RICHARD, TOPEKA
MELEAN, JAIME, WICHITA
MELENDRES, JUANITO M, OLATHE
MELHORN, J MARK, WICHITA
MELHORN MD, KATHERINE J, WICHITA
MENAKER, JEROME S, WICHITA
MENDIONES, L MARLENE, WICHITA
MENDIONES, RUPERTO O, WICHITA
MENDLICK, R MICHAEL, OLATHE
MENEHAN, H JAMES, WICHITA
MENEZ, CESAR V, SHAWNEE MISSION
MENKING, F W MANFRED, WICHITA
MENKING, SUSAN MARGARET, WICHITA
MENNINGER, KARL A, TOPEKA
MENNINGER, ROBERT G, TOPEKA
MENNINGER, ROY W, TOPEKA
MENNINGER, W WALTER, TOPEKA
MERCADER, MARIO S, WICHITA
MEREDITH, W TOM, WICHITA
MERKEL, EARL O, RUSSELL
MERRITT, JOE P, WICHITA
MERRITT, W HENRY, LEAVENWORTH
MERSHON, JAMES C, WICHITA
MESINA, ROLANDO R, KANSAS CITY
MEYER, MARK E, KANSAS CITY
MEYER, O WARREN, TOPEKA
MEYER, WARREN E, WICHITA
MEYERS, STEPHEN, GARDEN CITY
MICHELBACH, ALBERT P, WICHITA
MIGUELINO, OLIVER M, EMPORIA
MIH, ALEXANDER, CHANUTE
MILFELD, DOUGLAS J, WICHITA
MILLER, ABRAHAM H, MANHATTAN
MILLER, CHARLES H, PARSONS
MILLER, CLYDE W, WICHITA
MILLER, DAVID PATERSON, WICHITA
MILLER, DEAN M, PARSONS
MILLER, DON E, WICHITA
MILLER, EARL E, PITTSBURG
MILLER, ELDEN V, SALINA
MILLER, FRANKLIN R, WINFIELD
MILLER, FREEMAN LANCE, SHAWNEE MISSION
MILLER, HERBERT C, NORFORD,CT
MILLER, LAWRENCE H, DERBY
MILLER, MONTE B, SCOTT AFB, IL
MILLER, PHILIP A, WICHITA
MILLER, ROBERT E, GARDEN CITY
MILLER, STEPHEN FRANCIS, PARSONS
MILLIGAN, DONALD B, OLATHE
MILLS, CHARLES D, WICHITA
MILLS, GEORGE QUINTON, WICHITA
MILLS, VERNON A, LEAVENWORTH
MILLS JR, PHILIP E, TOPEKA
MIMOSO, JOSE J, DODGE CITY

* Probationary members.

MINER JR, PHILIP B, KANSAS CITY
MINNICK, CHARLES V, JUNCTION CITY
MINNS, GAROLD O, WICHITA
MIRZA, MEDO, WICHITA
MISKEW, DON B W, SHAWNEE MISSION
MITCHELL, ALEX C, LAWRENCE
MITCHELL, JOHN C, SALINA
MITTLEMAN, FREDERICK S, TOPEKA
MITTS, ERNEST W, BONNER SPRINGS
MODDRELL, CAROL A, LAWRENCE
MODLIN, HERBERT C, TOPEKA
MOELLER, DONALD O, KANSAS CITY
MOFFAT, ROBERT E, SHAWNEE MISSION
MOHLER, JACK M, ABILENE
MOHR, SANDRA NELL, WICHITA
MONCKTON, LAURANCE A, LAWRENCE
MONTGOMERY, LLOYD DAN, HALSTEAD
MONTGOMERY, THOMAS ALLEN, SABETHA
MONTGOMERYSHORT, RUTH G, HALSTEAD
MOORE, DENNIS F, WICHITA
MOORE, HUGH C, TOPEKA
MOORE, JAMES E, CONCORDIA
MOORE, JANE A, WICHITA
MOORE, ROBERT, HOISINGTON
MOORE, ROBERT F, CANEY
MOORE, WAYNE V, KANSAS CITY
MOORHEAD JR, F ALLEN, NEODESHA
MORALES, AMALIA D, OSAWATOMIE
MORALES, OTTO E, OSAWATOMIE
MORFFI, RAUL R, KANSAS CITY
MORGAN, DICK A, WICHITA
MORGAN, JAMES I, WICHITA
MORGAN, JOHN L, EMPORIA
MORGAN, NOVA L, HAYSVILLE
MORGAN II, DAVID LLOYD, OLATHE
MORGAN III, LOUIS S, WICHITA
MORONEY, JEAN M, SHAWNEE MISSION
MORRIS, MERLE O, TOPEKA
MORRISON, IRA R, ATCHISON
MORRISON, MICHAEL R, TOPEKA
MORRISON, RICHARD L, WICHITA
MORROW, THOMAS F, WICHITA
MORROW JR, J TARLTON, TOPEKA
MORTON, JOHN E, HALSTEAD
MOSELEY, JACK E, WICHITA
MOSER, ERNEST C, HOLTON
MOSER, M ROSS, HOLTON
MOSER, ROY H, HOLTON
MOSIER, MICHAEL L, MANHATTAN
MOSIER, STANLEY JAY, WICHITA
MOSIER, STEVEN J, MANHATTAN
MOWERY, WILLIAM E, SALINA
MOWRY, GERALD L, MANHATTAN
MOYER, HERMAN J, DERBY
MUEHLBERGER, JAMES J, SHAWNEE MISSION
MUELLER, ARNOLD V, TOPEKA
MUELLER, J KENT, SHAWNEE MISSION
MUELLER, VERNETTE A, WICHITA
*MUETH, JOAN O, WICHITA
MULL, JOHN C, HUTCHINSON
MULLEN JR, CLIFFORD J, KANSAS CITY
MULLEN SR, CLIFFORD J, KANSAS CITY
MULLER, SAMUEL B, PITTSBURG
MULLINIX, JANICE M, WICHITA
MURFITT, MALCOLM C, LINDSBORG
MURPHY, BARRY L, WICHITA
MURPHY, DUANE A, WICHITA
MURPHY, JAY W, SHAWNEE MISSION
MURPHY, JOHN F, KANSAS CITY
MURPHY, PAUL M, WICHITA
MURPHY, THOMAS MEAD, TOPEKA
MURRAY, KENT B, WICHITA
MURRAY, W LEE, SHAWNEE MISSION
MYERS, JO ANN, TOPEKA
MYERS, ROBERT W, NEWTON
MYERS, W EUGENE, IOLA
MYERS JR, EARL B, INDEPENDENCE
MYRICK, MICKEY, HAYS

N

NABOURS, RICHARD O, TOPEKA
NACHTIGALL, ANDREW, NEWTON
NAIK, GOPAL V, PAOLA
NALOOZA JR, FAUSTINO M, WELLINGTON
NANCE, JOEL, TOPEKA
NARCISO, VICENTE O, ABILENE
NASH, NEWMAN CURTIS, SCOTTSDALE,AZ
NASH, ROBERT A, LAWRENCE
NAUER, PAULA LOU, SHAWNEE MISSION
NAVICKAS, LEONARD A, SHAWNEE MISSION
NEEL, WILBUR B, HUTCHINSON
NEFF, JAMES R, KANSAS CITY
NEIBURGER, JAMES B, SHAWNEE MISSION
NEIGHBOR, ERNEST G, KANSAS CITY
NEIGHBOR, ERNEST H, KANSAS CITY
NEIGHBOR, GAYLORD P, KANSAS CITY
NEIGHBOR, RALPH M, EMPORIA
NEIL, ROY NEWTON, HAYS
NEIS, HARRY B, OSAGE BEACH,MO
NELSON, BRYAN C, SHAWNEE MISSION
NELSON, GERALD O, WICHITA
NELSON, JOHN B, SHAWNEE MISSION
NELSON, PAUL L, CONCORDIA
NELSON, RICHARD O, LAWRENCE
NELSON, RUSSELL ALAN, WICHITA
NELSON, T EUGENE, FORT SCOTT
NELSON, WILLIAM PAUL, KANSAS CITY
NELSON JR, GUST H, WICHITA

NEMMERS, DAVID J, WINFIELD
NESMITH, LESLIE W, WICHITA
NEUENSCHWANDER, JOHN, HOXIE
NEUENSCHWANDER, JOHN RAND, HOXIE
NEUER, FREDERICK S, EMPORIA
NEUSCHAFER, DARREL R, HUTCHINSON
NEVINS, RICHARD L, LIBERAL
NEVITT, J RUSSELL, MORAN
NEWBY, JAMES P, WICHITA
NEWCOMB, WARD M, HAYS
NEWKIRK, DARREL O, KANSAS CITY
NEWMAN, CARL T, CONCORDIA
NEWMAN, CLIFFORD B, PITTSBURG
NEWSOM, F CARTER, WICHITA
NEWTON, CHARLES R, VISALIA,CA
NIBBELINK, LARRY WAYNE, KANSAS CITY
NICE, G WILLIAM, TOPEKA
NICHOLS, RICHARD, COFFEYVILLE
NICHOLS, ROBERT R, FORT SCOTT
NICKELL, WAITSTILL B, SALINA
NICKELL, WENDELL K, SALINA
NIEDEREE, W CURTIS, GREAT BEND
NIELSEN, MARY L, HAYS
NIEMAN, JOHN L, SHAWNEE MISSION
NIENSTEDT, JOHN F, SUN CITY,AZ
NIKON, RICHARD R, CONCORDIA
NIXON, WILLIAM A, WICHITA
NOBLE, MARK J, KANSAS CITY
NOHE, PHILIP C, KANSAS CITY
NORRIS, CHARLEY W, KANSAS CITY
NORRIS, ROBERT P, WICHITA
NORTH, DORIS G, WICHITA
NORTH, VICTOR, WICHITA
NORTON, ROBERT K, WICHITA
NOSTI, JUAN C, SHAWNEE MISSION
NOTHNAGEL, ARNOLD F, KANSAS CITY
NOVOTNY, PETER C, TOPEKA
NOWLIN, NANCY S, WICHITA
*NUILA, RICHARD F, WICHITA
NULL, WILLIAM G, SALINA
NUNEMAKER, MARION E, HUTCHINSON
NUNEZ, JULIAN, KANSAS CITY
NYBERG, FREDRIK F, WICHITA
NYE, C ERIK, SHAWNEE MISSION
NYSTROM, CURTIS A, TOPEKA

O

O'BOYNICK II, PAUL LEONARD, KANSAS CITY
O'BRYAN, JAMES J, SHAWNEE MISSION
O'CONNELL, FRANK A, SHAWNEE MISSION
O'CONNELL, HAROLD F, ELLSWORTH
O'DONNELL, HARRY E, JUNCTION CITY
O'DONNELL, RICHARD H, CLAY CENTER
O'DONNELL JR, LEONARD A, WICHITA
O'DONNELL SR, LEONARD A, WICHITA
O'NEIL, ROBERT H, TOPEKA
O'SHEA, JAMES G, JETMORE
O'TOOLE, JAMES K, NEWTON
OBANDO, GUILLERMO, SALINA
OBOURN, ROBERT L, TOPEKA
OCHSNER, BRUCE B, WICHITA
OOENHEIMER, BURTRAM J, WICHITA
OOGERS, ROONEY K, PITTSBURG
OELSCHLAGER, RONALD O, LAWRENCE
OHMAN, RICHARD J, DODGE CITY
OHMART, RICHARD V, OAKLEY
OKTAWIEC, DANUTA, SHAWNEE MISSION
OLD, JERRY L, ARKANSAS CITY
OLOFIELD, RAY W, KANSAS CITY
OLNEY, ROBERT D, MANHATTAN
OLSEN, PHILLIP S, EL DORADO
*OLSON, BARBARA J, WICHITA
OLSON, CARL E, LAWRENCE
OLSON, CLITUS W, GOODLAND
OLSON, ERWIN T, NEWTON
OPENSHAW, CALVIN R, HUTCHINSON
ORCHARD, RICHARD A, LAWRENCE
OSBORN OD, ROBERT M, INDEPENDENCE
OSBORNE, CONRAD C, WICHITA
OSGOOD, GEORGE M, SHAWNEE MISSION
OSIO, ANTONIO L, WICHITA
OSOBA, WILLIAM G, WICHITA
OTHMER, EKKEHARD, KANSAS CITY
OUANO JR, BIBIANO B, MCPHERSON
OVERFIELD, A SCOTT, KANSAS CITY
OVERHOLSER, NORMAN H, EL DORADO
OWEN, GARRY D, LAWRENCE
OWEN, LARUE W, WICHITA
OWEN, PERE A, WICHITA
OWENS, RICHARD L, KANSAS CITY,MO
OWENSBY, L C, CONCORDIA
OKLER JR, JOHN EDWARD, KANSAS CITY

P

PACE, JOHN O, PARSONS
PADGETT, WILLIAM L, WICHITA
PAGE, O VALE, PLAINVILLE
PAGE, RUTH, WICHITA
PAI, RADHA V, PARSONS
PAI, VARADARAJ S, PARSONS
PAINE, GEORGE E, HUTCHINSON
PALMBERG, KENT E, TOPEKA
PALMER, DAVID L, WICHITA
PALMER, GERALD K, SALINA
PALMER, MARGUERITE L, OBERLIN

PALMER, MARVIN M, KANSAS CITY
PALMER JR, H C, LIBERAL
PALMERI, MARIA, KANSAS CITY
PALMERI, V CARLOS, KANSAS CITY
PANICHABHONGSE, SAMBHUNDH, RUSSELL
PAPP JR, S DEAN, PITTSBURG
PARAMESH, JAYA, LARNED
PARANJOTHI, SUBRAMONIAM P, PARSONS
PARDO, LILLIAN G, KANSAS CITY,MO
PARDO, MANUEL P, KANSAS CITY
PAREKH, AJITKUMAR M, KANSAS CITY
PARHAM, VERDON W, CHANUTE
PARK, CHAN H, KANSAS CITY
PARK, ROGER WALTER, WICHITA
PARKER, HAROLD L, WICHITA
PARKER, ROBERT W, LEAVENWORTH
PARKER, WAYNE G, EMPORIA
PARMAN, ROBERT O, TOPEKA
PARRA, MIGUEL O, KANSAS CITY
PARSI, MANJTCHEHR K, PITTSBURG
PARULKAR, DEEPAK S, TOPEKA
PASCHINO, ANTONIO R, KANSAS CITY
PASIMIO, ROGER S, COLUMBUS
PASTOR, VICTOR HUGO, EMPORIA
PATEL, MAHENDRA N, JUNCTION CITY
PATEL, VINOD, TOPEKA
PATINO, EDGAR, TOPEKA
PATRICK, FRED EDWARD, TOPEKA
PATTERSON, BRUCE W, WICHITA
PATTERSON, JOHN R, SHAWNEE MISSION
*PATTON, J MICHAEL, WICHITA
PAULS, DANIEL N, PARSONS
PAY, NORMAN T, WICHITA
PAYNE, J RALPH, KANSAS CITY,MO
PAYNE, ROBERT R, TOPEKA
PAZELL, JOHN A, KANSAS CITY
PEARCE, EUGENE W J, SHAWNEE MISSION
PEARCE, LUNETTA M, SHAWNEE MISSION
PEASE, GARY L, HUTCHINSON
PECANA, MANUEL C, KANSAS CITY
PEDERSON, ARNOLD M, PLAINVILLE
PEORAZA, HERNANDO, WELLINGTON
PEES, GERALD B, IOLA
PEES JR, GERALD BOYD, LAWRENCE
PEFFLY, ELMER O, CHETOPA
PENCE, CHARLES D, WICHITA
PENN, GEORGE M, TOPEKA
PENNINGTON, KATHERINE, WICHITA
PENTECOST, RICHARD L, SHAWNEE MISSION
PEROUE II, W LANG, TOPEKA
PEREIRA, WILLY G, ARKANSAS CITY
PERICO, CARLOS J, SHAWNEE MISSION
PERIOO, DOMINADOR T, ELKHART
PERKINS, JACK L, HUTCHINSON
PERRY JR, LAWRENCE L, KANSAS CITY
PETELIN, JOSEPH B, SHAWNEE MISSION
PETERIE, JERRY P, WICHITA
PETERS, GLENN R, KANSAS CITY
PETERS, THOMAS J, WICHITA
PETERSEN, A GENE, SHAWNEE MISSION
PETERSEN, GERALD D, SHAWNEE MISSION
PETERSON, DEAN L, TOPEKA
PETERSON, JACK T, MANHATTAN
PETERSON, ROBERT L, TOPEKA
PETERSON, VERNON J, TOPEKA
PETERSON JR, EVAN A, WATHENA
PETIT, CARL ALFONSO, SHAWNEE MISSION
PETRIE, SAMUEL C, SHAWNEE MISSION
PETRIK, EDWIN L, TOPEKA
PETTERSON, CECIL E, SYRACUSE
PETTERSON, DENNIS CRAIG, TOPEKA
PETTERSON, D'RUTH S, RIDGEVILLE,IN
PETTIJOHN, WALTER J, RUSSELL
PFUETZE, BRUCE L, SHAWNEE MISSION
PFUETZE, KARL D, SHAWNEE MISSION
PFUETZE, ROBERT E, TOPEKA
PHAM, TOAI KE, MONTEZUMA
*PHAN, DUNG MY, WICHITA
PHELPS, DAVID WAYNE, FORT SCOTT
PHILGREEN, DONALD E, OTTAWA
PHILIPP, JOSEPH THEODORE, MANHATTAN
PHILLIPS, STEPHEN B, MANHATTAN
PHILLIPS, WARREN G, SHAWNEE MISSION
PHIPPS, JACK G, WICHITA
*PICKENS, ANDREW T, WICHITA
PIERCE, CHARLES F, TOPEKA
PIERCE, DONALD R, TOPEKA
PIERCE, GEORGE E, KANSAS CITY
PIERRON, GEORGE J, OLATHE
PIERSON, WEIR, MCPHERSON
PILCHARD, WILLIAM A, SHAWNEE MISSION
PINCOMB, ARTHUR L, OLATHE
PINGLETON, WILLIAM WARREN, SHAWNEE MISSION
PINSKER, JACOB A, WICHITA
PISCHKE, FRANK J, KANSAS CITY
PITMAN, WILL D, PRATT
PITTS, RONALD L, SHAWNEE MISSION
PLOWMAN, CARL W, JEWELL
POGSON, GEORGE W, PITTSBURG
POL, P ALBERT, KANSAS CITY
POLING, TERRY L, WICHITA
POLLACK, SIMON, WICHITA
POLLAND DO, STEPHEN M, WICHITA
POLLOCK, ANTHONY G A, WICHITA
POLLY, RICHARD E, TOPEKA
POLSON, ROBERT C, GREAT BEND
POWWERENKE, FORREST A, DE SOTO
POOLE, BERNARD T, WICHITA
POONAWALA, HUSENI E, KANSAS CITY,MO

* Probationary members.

PORTER, DAVID M, KANSAS CITY
PORTER, GARRY L, WICHITA
PORTER, ROBERT D, TOPEKA
POTTER, ROBERT L, KANSAS CITY
POWELL, CAROL W, SHAWNEE MISSION
POWELL, KENNETH A, SHAWNEE MISSION
POWELL, WILLIAM R, TOPEKA
POWELL II, BENSON M, TOPEKA
POWERS, G ROBERT, KANSAS CITY
POWERS, HAROLD W, SUN CITY,AZ
POWERS, K DEAN, WICHITA
PRAEGER, MARK A, LAWRENCE
PRAKALAPAKORN, DARANEE, NESS CITY
PRAKALAPAKORN, YANYONG, NESS CITY
PRAY, CLAUDIA M, LEAVENWORTH
PREHEIM, OELBERT V, NEWTON
PREMSINGH, NALINI G, KANSAS CITY
PRENDES, CARLOS A, SHAWNEE MISSION
PRENTISS, HAROLD, NEWTON
PRESKORN, SHELDON H, KANSAS CITY
PRESTON, DAVID F, KANSAS CITY
PRESTON, RALPH R, TOPEKA
PRESTON, RICHARD, GREAT BEND
PRETZ, JAMES B, KANSAS CITY
PRICE, HILTON I, KANSAS CITY
PRICE, JAMES GORDON, KANSAS CITY
PRICE, VAUGHAN C, MCPHERSON
PRICE JR, LAURANCE W, LAWRENCE
PRIETO, JORGE N, KANSAS CITY
PRIETO, LUIS E, WICHITA
PROCHASKA, MARK L, KANSAS CITY
PROCHAZKA, OTTO F, LIBERAL
PROCTOR, ROBERT W, EL DORADO
PROKOP, BRADFORD S, TOPEKA
PRONKO, MICHAEL J, SHAWNEE MISSION
PROUD, G ONEIL, KANSAS CITY
PUGH, DAVID M, KANSAS CITY
PULLMAN, NORMAN K, WICHITA
*PULLUM, RICHARD W, WICHITA
PURINTON, LEW W, WICHITA
PUTNAM, LYLE B, WICHITA
PYLE, LUCIEN R, TOPEKA

Q

QAMAR, YUSUF, NEWTON
QUACKENBUSH, ROBERT P, SPEARVILLE
QUENZER, RONALD W, PRATT
QUINN, CHARLES E, KANSAS CITY
QUINONES, ELADIO A, TAMPA,FL

R

RABE, MELVIN A, LEAVENWORTH
RACELA JR, ANTONIO S, SHAWNEE MISSION
RADOVANOV, RADMILA, NEWTON
*RAGHAVAN, PARULA P, WICHITA
RAGHAVAN, PRAKASH V, WICHITA
RAGLAND, CHARLES W, KANSAS CITY
RAHMAN, HAFIZ M A, TAMPA,FL
RAINBOW-EARHART, KATHRYN A, TOPEKA
RAJEWSKI, RICHARD L, HAYS
RAJU, A S PADMA, TOPEKA
RALSTIN, JAMES H, SHAWNEE MISSION
RAMCHANDANI, A P, ULYSSES
RAMIREZ, AUGUSTO H, PITTSBURG
RAMIREZ, IRENE, PITTSBURG
RAMSEY, BARTLETT W, TOPEKA
RANDALL, GEORGE R, WICHITA
RANOLES, MICHAEL J, WICHITA
RANSOELL, EDGAR C, TOPEKA
RANSOM, JAMES H, TOPEKA
RANSOM, KENNETH J, WICHITA
RANSOM, WILLARD B, OTTAWA
RAROIN, MARY L, WICHITA
RASSA, REZA P, SALINA
RATE, PEGGY S, HUTCHINSON
RATE, ROBERT R, HUTCHINSON
RATHBUN, EDWIN D, LIBERAL
RAUSA JR, FRANCISCO C, WICHITA
RAVIPHANTHAKUK, KOBCHI, WICHITA
RAWCLIFFE JR, ROBERT A, WICHITA
RAZEK, ZACK A, WICHITA
READ, WILLIAM T, COFFEYVILLE
*READER, G WHITNEY, WICHITA
REALS, WILLIAM J, WICHITA
REAZIN, WALTER L, WICHITA
RECKLING, FREDERICK W, KANSAS CITY
REDDI, RAGHUNATH P, WICHITA
REDDY, B N, HILL CITY
REDDY, EASHWER K, KANSAS CITY
REDDY, P JAGANNAOHA, HILL CITY
REDDY, SATTI S, DODGE CITY
REDDY, VENUMBAKA C, EL DORADO
REDFORD, JOHN W B, KANSAS CITY
REDING, DOUGLAS J, KANSAS CITY,MO
REEB, RONALD JOSEPH, KANSAS CITY
REECE, A THOMEN, GARDNER
REECE, RICHARD J, SALINA
REED, A J, WICHITA
REED, D CRAMER, WICHITA
REED, DAVID O, WICHITA
REED, JAMES S, APO MIAMI,FL
REED, JAMES STEWART, KANSAS CITY
REED, RALPH R, LAWRENCE
REESE, JACK D, LIBERAL
REESE, JOHN L, LAWRENCE

REEVES, BRADFORD F, WICHITA
REEVES, CHARLES S, FORT SCOTT
REGIER, HENRY L, KANSAS CITY
REGIER, LADONNA M, COLBY
REILLY, DOUGLAS K, KANSAS CITY
REINHARDT-WULF, TAISSIA L, GARDEN PLAIN
REINKING, VICTOR E, TOPEKA
REISMAN, MICHAEL ALAN, WICHITA
REITH, PAUL, LAWRENCE
REITZ, LELAND C, MANHATTAN
REITZ, ROGER P, MANHATTAN
REIVICH, RONALD S, SHAWNEE MISSION
RELIHAN, DONALD A, WICHITA
RELIHAN, FRANCIS H, SMITH CENTER
REMPEL, JOHN H, WICHITA
RENOON, HUMBERTO M, AL AIN,
REPLOGLE, CHARLES B, GREAT BEND
RESCHLY, RON, HALSTEAD
REYES JR, FRANCISCO A, OTTAWA
REYMOND, RALPH D, TOPEKA
REYNOLDS, JEFFREY C, HAYS
REYNOLDS, LLOYD W, HAYS
RHOADS, JAMES P, WICHITA
RHODEN, CURTIS H, WICHITA
RHODES, IVAN E, WICHITA
RHODES, JAMES B, KANSAS CITY
RHODES, LOWELL M, WICHITA
RHODES, MARTIN L, KANSAS CITY
RICCI, ROBERT LAWLER, TOPEKA
RICE, BERNARD F, SHAWNEE MISSION
RICE JR, FREDERICK A, KANSAS CITY
RICH, ELDON S, NEWTON
RICH, JOSEPH E, TOPEKA
RICHARDS, DALLAS LEE, HAYS
RICHARDS, DENNIS D, CLAY CENTER
RICHARDS, JON F, PHILLIPSBURG
RICHARDSON, J M, TOPEKA
RICHARDSON, STEWART F, WICHITA
RICHTER, DON G, SHAWNEE MISSION
RICK JR, GREGORY G, SHAWNEE MISSION
RIDER, JAMES W, ATCHISON
RIEDEL, ROBERT H, TOPEKA
RIEDERER, ROBERT E, WICHITA
RIEGER, ERNEST H, WICHITA
RIEKE, FRANK A, SHAWNEE MISSION
RIEPE, ROGER E, WICHITA
RIFFEL, LAWRENCE D, SHAWNEE MISSION
RIGLER, WILSON F, ARMA
RILEY, RAY B, KANSAS CITY
RINOT, PHIL, FREDONIA
RINKENBERGER, ROBERT, KANSAS CITY
RIORDAN, HUGH D, WICHITA
RISING, JESSE D, KANSAS CITY
RITCHIE, KAREN S, SHAWNEE MISSION
RIZZA, ROBERT G, HALSTEAD
ROACH, NEIL E, WICHITA
ROBERTS, DANIEL K, WICHITA
ROBERTS, RICHARD S, LAWRENCE
ROBERTS, WARREN E, TOPEKA
ROBERTSON, EDWARD J, SHAWNEE MISSION
ROBERTSON, JOSEPH K, WICHITA
ROBINSON, DAVID B, TOPEKA
ROBINSON, DAVID W, KANSAS CITY
ROBINSON, EDGAR L, INDEPENDENCE
ROBINSON, G DONALD, WICHITA
ROBINSON, JOHN D, KANSAS CITY
ROBINSON, JOHN E, WICHITA
ROBINSON, MICHAEL O, HAYS
ROBINSON, RALPH G, KANSAS CITY
ROBINSON, ROBERT H, WICHITA
ROBISON, JAMES T, SHAWNEE MISSION
ROBL, DAVID A, WICHITA
ROCHANAYON, PIRA, ELLIS
RODERICK, JAMES E, SALINA
RODRIGUEZ, ALBERTO, KIOWA
RODRIGUEZ, PAUL L, GARDEN CITY
RODRIGUEZ, RAUL G, KANSAS CITY
*RODRIGUEZ-RAMOS, ERNEST R, WICHITA
RODRIGUEZTOCKER, LILIA, WICHITA
ROEDER, ROBERT E, TOPEKA
ROMALIS, BRIAN E, WICHITA
ROMEISER, REK S, SALINA
ROMITO, CYNTHIA L, SHAWNE MISSION
ROMONDO, STEVEN A, OLATHE
ROOK, LEE E, KANSAS CITY
*ROOS, MAUREEN, WICHITA
RORABAUGH, DONALD C, ABILENE
ROSE, DONALD L, BELLA VISTA,AR
ROSE, GRAHAM C, MANHATTAN
ROSE, SHELBY D, WICHITA
ROSEN, DAVID, WICHITA
ROSENBERG, STANTON L, SHAWNEE MISSION
ROSENBERG, THOMAS F, WICHITA
ROSENTHAL, RICHARD, SHAWNEE MISSION
ROSENTHAL, STANTON J, KANSAS CITY
ROSS, DAVID K, ARKANSAS CITY
ROSS, DENNIS LEE, WICHITA
ROSS, JACK L, TOPEKA
ROSSITTO, ANTHONY F, SAN FRANCISCO,CA
ROTERT, LARRY, TOPEKA
ROTH, ALAN E, KANSAS CITY
ROTHSTEIN, TERRY B, PARSONS
ROWLETT, JACK G, PAOLA
ROY, WILLIAM R, TOPEKA
RUBIN, HERBERT M, SHAWNEE MISSION
RUBIN JR, BEN, KANSAS CITY
RUBLE JR, JAMES L, OVERBROOK
RUCKER, CLEMENS, TOPEKA
RUEB, ANDREW E, SALINA
RUHLEN, JAMES L, OLATHE

RUIZ, CARLOS M, GREAT BEND
RUMOLD, MERVIN J, SHAWNEE MISSION
RUNNELS, JOHN B, TOPEKA
RUPP, RICHARD J, TOPEKA
RUSSELL, PHILIP W, WICHITA
RUTH, WILLIAM E, KANSAS CITY
RUTNGAMLUG, LUECHA, HAYS
RUZICKA, LAWRENCE J, CONCORDIA
RYAN, MICHAEL E, SHAWNEE MISSION
RYAN, MICHAEL J, KANSAS CITY
RYAN, W SCOTT, EMPORIA
RYMER, ROBERT A, SHAWNEE MISSION

S

SABIN JR, GEORGE M, WICHITA
SACHEN, FREDERICK L, SHAWNEE MISSION
SADIQ, SULEMAN, WICHITA
SAEED, MOHAMMAD, WICHITA
SAFFO, KARL S, SHAWNEE MISSION
SAMUEL, CHANDY C, WINFIELD
SAMUEL, SHANTHI, WINFIELD
SANCHEZ, ROGELIO, TOPEKA
SANDBERG, CHRIS B, EL DORADO
SANDERS, GLORIA DEANNA, WICHITA
SANDERS, J ALAN, LAWRENCE
SANDHU, PAUL S, COFFEYVILLE
SANTOSCOY, GILBERT S, WICHITA
SARGENT, JOSEPH D, TOPEKA
SATHYANARAYANA, SARASWATHI, SHAWNEE MISSION
SAUL, F WILLIAM, EMPORIA
SAUNDERS, MICHAEL E, CONCORDIA
SAVAGE, WILLIAM R, HUTCHINSON
SAVIN, VIRGINIA J, KANSAS CITY
SAWKAR, LAXMIDAS A, SHAWNEE MISSION
SAYLER, JEROME, GREAT BEND
SAYLOR, EDWARD H, TOPEKA
SAYLOR, LESLIE L, TOPEKA
SAYLOR, MARK, TOPEKA
SAYLOR, STEPHEN, TOPEKA
SCALES, WILLIAM M, HUTCHINSON
SCAMMAN, W WIKE, TOPEKA
SCANLAN, TIMOTHY M, WICHITA
SCHAEFER, JOSEPH PETER, SHAWNEE MISSION
SCHAFFER, CLARENCE K, SANTA CRUZ, CA
SCHEEL, BRADLEY J, HUTCHINSON
SCHELLINGER, RICHARD P, EMPORIA
SCHERER, ALFRED L, ST JOHN
SCHILTZ, FRANCES, WICHITA
SCHIMKE, R NEIL, KANSAS CITY
SCHLACHTER, ERNEST R, WICHITA
SCHLEMMER, ROGER B, PITTSBURG
SCHLICHER, JOHN E, WICHITA
SCHLOESSER, HARVEY L, TOPEKA
SCHLOESSER, PATRICIA T, TOPEKA
SCHLOTTERBACK, WILLIAM E, MANKATO
SCHLUETER, JOHN J, WICHITA
SCHMAUS, LYLE F, IOLA
SCHMIDT, HERBERT R, NEWTON
SCHMIOT, RAMON WARNER, SALINA
SCHNELLE, JOACHIM, WICHITA
SCHNDEBELEN, RENE E, KINSLEY
SCHNOSE, GREGORY O, LAWRENCE
SCHOPF, CLIFTON C, WICHITA
SCHOTLAND, EDWARD S, KANSAS CITY
SCHRAM, PETER CHARLES, TOPEKA
SCHREPFER, ROSEMARY, KANSAS CITY
SCHROEDER, SYDNEY O, LAWRENCE
SCHROLL, JACK C, HUTCHINSON
SCHROLL, JOHN T, SHAWNEE MISSION
SCHUETZ, PERRY N, GREAT BEND
SCHUKMAN, JAY S, GREAT BEND
SCHULTZ, JAMES E, COUNCIL GROVE
SCHWARTING, J STEVE, ABILENE
SCHWARTZ, EUGENE W, DODGE CITY
SCHWARTZ, V DEAN, WICHITA
SCHWEGLER, RAYMOND A, LAWRENCE
SCHWEGLER, RAYMOND A, KANSAS CITY
SCHWORM, CURTIS P, KANSAS CITY
SCLAR, WILLIAM C, SHAWNEE MISSION
SCOTT, ALEX, JUNCTION CITY
SCOTT, CHESTER E, SALINA
SCOTT, DUANE L, BELLEVILLE
SCOTT, VINCENT L, WICHITA
SCOTT, WILLIAM H, WICHITA
SEAGO, CHARLOTTE L, LIBERAL
SEAMAN, LAUREN I, OLATHE
SEBREE, STEVEN G, SALINA
SEELEY, JAMES C, HOLTON
SEGERSON, JOHN A, TOPEKA
SEGLIE, FLOYD RONALD, PITTSBURG
*SEGURA, ANTONIO, TOPEKA
SEHDEV, JOAN, TOPEKA
SEITZ JR, JOSEPH E, ELLSWORTH
SEKAVEC, GORDON B, OAKLEY
*SEN SARMA, PRONAB K, WICHITA
SERERES, EDGAR P, KANSAS CITY
SETTLE JR, RUSSELL O, OLATHE
SETTLE SR, RUSSELL O, TOPEKA
SEVIER, SAMUEL M, MUSKOGEE, OK
SHAAD, DOROTHY J, SHAWNEE MISSION
SHAFER, PRESTON J, WICHITA
SHAH, ASHOK H, WINFIELD
SHAH, MIAN, LARNED
SHAH, MUKHTAR H, WICHITA
SHAH, NASREEN, LARNED
SHAH, SHARFUDDIN, HALSTEAD

SHANKER, STUART G, OLATHE
SHARMA, ARUN L, PARSONS
SHAW, JOSEPH L, TOPEKA
SHAW, RICHARD C, WICHITA
SHAW JR, JAMES W, HUTCHINSON
SHEAFOR, DOUGLAS, TOPEKA
SHEARS, ROBERT N, HUTCHINSON
SHEERN, MARK DOUGLAS, ABILENE
SHEFFER, KEITH O, OLATHE
SHEIKH, MASOOD A, INDEPENDENCE
SHELLITO, JOHN G, WICHITA
SHELTON, STEPHEN E, TOPEKA
SHEPARD, LERDY W, LARNED
SHEPPARD, ROBERT G, SMITH CENTER
SHERIDAN, RANDY M, KANSAS CITY
SHERMAN, ROBERT P, KANSAS CITY
SHERWOOD JR, CLARENCE E, TOPEKA
*SHIELD, CHARLES, WICHITA
SHIELDS JR, JAMES M, EL DORADO
SHIFLET, ALBERT W, WHEELING, MD
SHIVEL, DAVID G, GREAT BEND
SHOFSTALL, WILLIAM H, SHAWNEE MISSION
SHORTES, LOIS E, OSWEGO
SHRADER, DOYLE A, WICHITA
*SHURTZ, GLEN L, WICHITA
SHUSS, JOHN L, GARDEN CITY
SIBALA, JUSTO L, PRATT
SIEGEL, ALBERT R, WICHITA
SIEMENS, RICHARD A, LYONS
SIFERS, EARL C, KANSAS CITY
SIFERS, TIMOTHY M, KANSAS CITY
SIFFORD, R LAWRENCE, WICHITA
SILER, EUGENE T, HAYS
SILLS, CHARLES T, NEWTON
SILLS, THOMAS D, SHAWNEE MISSION
SILVER, BRADO J, SHAWNEE MISSION
SILVERS, ALVIN, KANSAS CITY
SIMMONS, ROBERT EARLE, NEWTON
SIMPSON, J COLBERT, SALINA
SIMPSON, ROBERT LIMBAUGH, DBERLIN
SIMPSON, TOM C, STERLING
SIMPSON, WILLIAM S, TOPEKA
SINGER, PHILIP A, KANSAS CITY, MO
SINGH, GIRVAR, ARKANSAS CITY
SINNING, GARY, HIAWATHA
SISK, PHILLIP B, TOPEKA
SIWEK, CHRISTOPHER W, EL DORADO
SKAER, STANLEY ALLEN, EUREKA
SKIBBA, RICHARD M, WICHITA
SKIKNE, BARRY S, KANSAS CITY
SLEEPER, CAROL A, KANSAS CITY, MO
SLOO, MILO G, SALINA
SLUTSKY, LAWRENCE JOEL, WICHITA
SMITH, ALVIN L, WICHITA
SMITH, BOYD E, SALINA
SMITH, BRUCE G, ARKANSAS CITY
SMITH, DALE C, SHAWNEE MISSION
SMITH, DAVID E, SALINA
SMITH, DONALD J, SHAWNEE MISSION
SMITH, HAROLD R, SALINA
SMITH, JOHN D, LARNED
SMITH, LEO A, TOPEKA
SMITH, NEWTON C, ARKANSAS CITY
SMITH, PERRY MILTON, GREAT BEND
SMITH, STEPHEN D, KANSAS CITY
SMITH, THOMAS WILLIAM, HUTCHINSON
SMITH, TIMOTHY WM, WICHITA
SMITH, WILLIAM P, KANSAS CITY, MO
SMITH JR, FLOYD L, COLBY
SMITH JR, WILLARD J, WICHITA
SNARR, JACK W, TOPEKA
SNODELL, FIRMIN E, SHAWNEE MISSION
SNOOGRASS, WAYNE R, KANSAS CITY
SNOOK, ROBERT RUFUS, MCLOUTH
SNOW, DONALD L, LANSING
SNOW JR, ARTHUR O, SHAWNEE MISSION
SNOWBARGER, MARVIN D, EMPORIA
SNYDER, C JOHN, WINFIELD
SNYDER, GREGG M, WICHITA
SNYDER, HOWARD E, WINFIELD
SNYDER JR, RICHARD HENRY, OLATHE
SOELDNER, JAMES OLIVER, SHAWNEE MISSION
SOLOMON, HERMAN, WICHITA
SOLTZ, ROBERT A, WICHITA
SOMERS, MARVIN M, WICHITA
SONGER, HERBERT L, ABILENE
SOUCEK, CHARLES O, KANSAS CITY
SOUTHWICK, CHRISTOPHER L, EDWARDSVILLE
SPAKE, ROBERT V, KANSAS CITY
SPANN, RICHARD W, WICHITA
SPAULDING, JOHN S, KANSAS CITY
SPEARMAN, JESSE L, TOPEKA
SPEARS, CHESTER A, EMPORIA
SPEER, FREDERIC, SHAWNEE MISSION
SPEER, LELAND, KANSAS CITY
SPEER, LOJIS N, OTTAWA
SPENCER, JOHN HAROLD, FORT SCOTT
SPENCER, MILLARD C, TOPEKA
SPENCER, WAYNE E, TOPEKA
SPIKES, MARION E, GARDEN CITY
SPITZER, JEROME S, HUTCHINSON
STADALMAN, ROSS EUGENE, HAYS
STAFFORD, ROBERT W, HUTCHINSON
STANLEY, KENNETH E, WICHITA
STANLEY, REX C, PAOLA
STARK, JAMES R, WICHITA
STARKEY, JERALD L, RUSSELL
STASS-ISERN, MERRILL, KANSAS CITY, MO
STECH, JOSEPH M, ANDALE
STECHSCHULTE, DANIEL J, KANSAS CITY

STECKLEY, RICHARD ALLEN, WICHITA
STEEGMANN, A THEODORE, INDIANAPOLIS, IN
STEELE, CLARENCE H, KANSAS CITY
STEICHEN, EDWARD F, LENORA
STEIN, JOSEPH M, TOPEKA
STEIN, PAUL S, WICHITA
STEINBAUER, JEFFREY ROBERT, CLAY CENTER
*STEINER, FREDERICK T, TOPEKA
STEINER, ROBERT M, SHAWNEE MISSION
STEINKRUGER, VERLYN WILLIAM, SMITH CENTER
STEINMETZ, WM, HUTCHINSON
STEINZEIG, ALFRED S, SHAWNEE MISSION
STEINZEIG, SHERMAN M, KANSAS CITY
STEMBRIDGE, TRAVIS W, WICHITA
STENSAAS, CARL O, HUTCHINSON
STEPHANZ JR, GERALD B, WICHITA
STEPHENS, CHARLES, MINNEOLA
STEPHENS, RONALD L, KANSAS CITY
STEPHENSON, LUCILLE C, ST FRANCIS
STEVENS, MILDRED J, GARNETT
STEVENS, PHILIP L, TONGANOXIE
STEVENS, ROBERT L, GARNETT
STEVENSON, E KENT, SHAWNEE MISSION
STILLIE OO, G DONALD, KANSAS CITY, MO
STITES III, HAROLD W, OLATHE
STITT, RONALD W, SHAWNEE MISSION
STIVERS, MARK T, LAWRENCE
STOCK, KARL W, TOPEKA
STOCKWELL, MORGAN U, DODGE CITY
STOFER, BERT E, PRESCOTT, AZ
STOFFER, ROBERT P, HALSTEAD
STOKES, ROBERT LEE, KANSAS CITY, MO
STOLZ, ELMER G, WICHITA
STONE, G REX, MANHATTAN
STONE, GRANT C, ATTICA
STOSKOPF, LAWRENCE E, SALINA
STOUT, JAMES M, HUTCHINSON
STOUT, NILES M, LYNDON
STREET, DAVID E, WICHITA
STREHLOW, CHESTER H, OTTAWA
STREIT, JEROME G, WICHITA
STRICKLAND, MAJRICE VAN, WICHITA
STRIEBINGER, CHARLES M, SHAWNEE MISSION
STRUTZ, WILLIAM C, LEAVENWORTH
STRYKER, TERRY MARGARET, WICHITA
STRYKER JR, HENRY B, CONCORDIA
STUBBLEFIELD, CHARLES T, KANSAS CITY
STUBER, JACK LAWRENCE, SHAWNEE MISSION
STUCKEY, CHARLES E, SHAWNEE MISSION
STUCKY, DEAN E, MEDICINE LODGE
STUEWE, BRAD R, SALINA
STUMP, HARL G, HAYS
SUERO, JESUS T, WICHITA
SUFI, M ASHRAF, TOPEKA
SUGAR, ROBERT L, SHAWNEE MISSION
SUITER, DANIEL JAY, PRATT
SULLIVAN, CORNELIUS J P, WICHITA
SULLIVAN, LEONARD L, WICHITA
SULLIVAN, TOM G, SHAWNEE MISSION
SULLIVAN JR, HENRY B, SHAWNEE MISSION
SUMNER, JOYCE R, HUTCHINSON
SUMNER, MARION M, HUTCHINSON
SUMNER, RALPH N, FREDONIA
SURFACE, GARDNER A, ELLIS
SUTTON, RICHARD O, TOPEKA
SUTTON, ROBERT E, INDEPENDENCE
SUTTON JR, RICHARD L, SHAWNEE MISSION
SVOBODA, CHARLES R, CHAPMAN
*SVOBODA, LOIS V, WICHITA
*SVOBODA, WILLIAM B, WICHITA
SWAN, MAJOR MARTIN, GREAT BEND
SWANN, CLAIR L, RUSSELL
SWARTZ, WARREN E, PARSONS
SWISHER, WILLIAM C, WICHITA
SWOGGER JR, GLENN, TOPEKA
SZYMANSKI, DENNIS C, SHAWNEE MISSION

T

TADURAN, VIRGILIO, SATANTA
TALLEY, ROBERT L, SHAWNEE MISSION
TAMBURINI, MARIO, LEAVENWORTH
TANDOC JR, VALENTIN T, NEWTON
TANGCHUPONG, SAROHO, PARSONS
TAPPEN, DANIEL L, TOPEKA
TARGOWNIK, KARL K, TOPEKA
TARNOFF, GERALD M, TOPEKA
TARNOWER, WILLIAM, TOPEKA
TATPATI, DANIEL A, WICHITA
TATPATI, OLGA ADELINA, WICHITA
TAYIEM, ABDEL K, ATCHISON
TAYLOR, BARBARA O, MANHATTAN
TAYLOR, E J, HUTCHINSON
TAYLOR, ELMER W, SEDAN
TAYLOR, RICHARD J, WICHITA
TAYLOR, SARAH A, KANSAS CITY
TAYLOR, STEVEN L, WICHITA
TAYLOR, THOMAS F, SALINA
TAYLOR, THOMAS L, KANSAS CITY
TEARE, MAX E, GARDEN CITY
TEJANO, NEONILO A, HALSTEAD
TEMPERO, STEPHEN J, TOPEKA
TEMPLETON, ARCH W, KANSAS CITY
TENNY, ROBERT T, SHAWNEE MISSION
TETZLAFF, ARCHIBALD O A, SHAWNEE MISSION
THELEN, J CHRISTINE, WICHITA
THEROU, LEONA F, KANSAS CITY
THIEMANN, A H, O.D., SUBLETTE
THOMAS, GREGORY MCQUEEN, MCPHERSON

* Probationary members.

THOMAS, JAMES H, KANSAS CITY
THOMAS, R CULLEN, PRATT
THOMAS, THOMAS V, KANSAS CITY
THOMPSON, DANIEL M, WICHITA
THOMPSON, DANNIE M, KANSAS CITY
THOMPSON, SCOTT W, WICHITA
THOMS, NORMAN W, TOPEKA
THOMSEN, GARY, SHAWNEE MISSION
THOMSEN, STEVEN T, LAWRENCE
THORNTON JR, FOXHALL P, CONCORDIA
THORPE, FRANCIS A, PRATT
THURSTON, DAVID E, TOPEKA
TIEMANN, WILLIAM H, MANHATTAN
TIETZE, DENNIS O, TOPEKA
TIHEN, EDWARD N, WICHITA
TIHEN, HENRY N, WICHITA
TILLER, GEORGE R, WICHITA
TILLOTSON, DON R, ULYSSES
TILTON, FRANK M, WICHITA
TINKER, ROBERT C, WICHITA
TINTEROW, MAURICE M, WICHITA
TIPPIN JR, ERNEST E, WICHITA
TISDALE, TERRANCE C, HUTCHINSON
TIVORSAK, ARKOM, ATCHISON
TOALSON, WILLIAM B, SHAWNEE MISSION
TOCKER, ALFRED M, WICHITA
TOMPKINS, CARL O, NEWTON
TONN, GERHART R, WICHITA
TOSH, FRED E, WICHITA
TOSONE, STEVEN R, KANSAS CITY
TOTH, JOHN ROY, TOPEKA
TOUT, ROBERT C, MANHATTAN
TOZER, RICHARD C, TOPEKA
TRACY, TERRY A, WICHITA
TRAVIS, JOHN W, TOPEKA
TREES, CLYDE B, TOPEKA
TREES, DONALD P, HOLLYWOOD,CA
TREGER, NEWMAN V, TOPEKA
TREKELL, WILLIAM V, DODGE CITY
TRETBAR, HARVEY A, WICHITA
TRETBAR, LAWRENCE L, SHAWNEE MISSION
TREWEEKE, MICHAEL W, WICHITA
TRIMBLE SR, DAVID P, EMPORIA
TROPP DO, ARNOLD L, SHAWNEE MISSION
TROTTER, ROGER COURTNEY, DODGE CITY
TRUDEAU, DAVID L, WICHITA
TRUEWORTHY, ROBERT C, KANSAS CITY
*TRUJILLO, ANTERO A, WICHITA
TUCKER, DONALD R, LAWRENCE
TUCKER, SHERIDAN G, SHAWNEE MISSION
TUCKER, VIRGINIA L, TOPEKA
TUONG, TRAN MANH, AUGUSTA
TURNER, JOHN W, GARDEN CITY
TUTERA, GINO, SHAWNEE MISSION
TWEET, FREDRICK A, PITTSBURG
TWEITD, DAVID H, HUTCHINSON
*TWEMLOW, STUART W, TOPEKA

U

UBELAKER, ERNEST J, SOUTH HAVEN
UHLIG, PAUL J, WICHITA
UHR, NATHANIEL, TOPEKA
*ULRICH, BRIAN K, WICHITA
UNDERWOOD, CHARLES C, EMPORIA
UNREIN, ROBERT J, GREAT BEND
UTLEY, JAMES HARMON, KANSAS CITY
UY, WILSON O, COFFEYVILLE

V

VACHAL, EVA, GARDEN CITY
VAKAS, JOHN L, COFFEYVILLE
VALK, WILLIAM L, SHAWNEE MISSION
VAN HOUDEN, CHARLES E, CHANUTE
VAN LEEUWEN, GERALD, WICHITA
VAN SICKLE, GREGGORY J, TOPEKA
VAN THULLENAR, PHILIP A, KANSAS CITY
VANDE GARDE, LARRY D, TOPEKA
VANDER VELDE, STANLEY L, EMPORIA
VANNAMAN, DONALD D, SHAWNEE MISSION
VARGHESE, GEORGE, KANSAS CITY
VATS, TRIBHAWAN S, KANSAS CITY
VAUGHAN, DONNA A, NEWTON
VELARDE, HUGO, KANSAS CITY
VERMA, ASHA, PARSONS
VERNON, MARY C, LAWRENCE
VIERTHALER, CARL A, DODGE CITY
VILLANUEVA, CESAR L, OLATHE
VIN ZANT, LARRY E, WICHITA
VINE, DONALD LEE, WICHITA
VINZANT, MARK N, DERBY
VINZANT, WHITNEY L, WICHITA
VISSER, VALYA E, KANSAS CITY
VITALE, NEIL B, KANSAS CITY,MO
VOGEL, STANLEY J, TOPEKA
VOGT, VERNON W, NEWTON
VOLKMANN II, HARLEY W, MANHATTAN
VON GKASEMSIRI, SUNAN, NEWTON
VON LEONROD JR, GEORGE, DIGHTON
VON RUDEN, WILLIAM J, HUTCHINSON
VOORHEES, CARROLL O, LEAVENWORTH
VOORHEES, GORDON S, LEAVENWORTH
VORHEES, VICTOR J, WICHITA
VOTAPKA, WILLIAM L, STOCKTON
VOTH, DOUGLAS W, WICHITA
VOTH, HAROLD M, TOPEKA

W

WADDELL, BILL D, KANSAS CITY
WADE, THEODORE E, LIBERAL
WADE III OD, WILLIAM E, TOPEKA
WADUD, ABDUL, WICHITA
WAGENBLAST, HOWARD R, SALINA
WAGGONER, FRANKLIN E, BONNER SPRINGS
WALDORF JR, MELVIN H, GREENSBURG
WALKER, JACK O, KANSAS CITY
WALKER, MAURICE A, KANSAS CITY
WALKER, NELLIE G, LEES SUMMIT,MO
WALKER, WILLIAM H, ESKRIDGE
WALKER, WILLIAM K, SEDAN
WALKER, WILLIAM L, SHAWNEE MISSION
WALLACE, LEO F, TOPEKA
WALLACE JR, WAYNE O, ATCHISON
WALLS, WILLIAM J, TOPEKA
WALSH, THOMAS E, ONAGA
WALTER, DONNAL C, KANSAS CITY
WALTERS, BYRON W, SUN CITY,AZ
WALTERS, ROBERT MERRILL, WICHITA
WALTERS, WILLIAM DAVID, KANSAS CITY
WALTON, PHILIP D, HORTON
WALZ, ROYCE C, TOPEKA
WALZ, THOMAS J, ST FRANCIS
WANG, SIDNEY W, SHAWNEE MISSION
WANLESS, KIRK M, TOPEKA
WARD, HOWARD N, TOPEKA
WARD, JAMES A, BELLEVILLE
*WARD, L G, WICHITA
WARD, ROBERT L, STAFFORD
WARE, LUCILE M, TOPEKA
WARREN, LINDA D, HANOVER
WARREN, LLOYD P, WICHITA
WARREN, ROGER D, HANOVER
WARREN, WIRT A, WICHITA
WARREN JR, JOHN W, WICHITA
WARRICK, DAVID ALAN, TOPEKA
WATERS, CLARENCE N, SALINA
WATERS, DALE A, TOPEKA
WATTS, HARRY E, HAYS
WAXMAN, DAVID, KANSAS CITY
WAYLAN, THORNTON L, NASHVILLE
WEAVER, J ROBERT, WICHITA
WEAVER, JACK D, WICHITA
WEAVER, WALTER D, TOPEKA
WEBB, JAMES R, SHAWNEE MISSION
WEBER, DARRELL J, TOPEKA
WEBER, ROBERT W, SALINA
WEBER, ROY R, NEWTON
WEBER, WALLACE N, HAYS
WEBER II, RALPH H, SALINA
WEBER JR, HUGO P, WICHITA
WEBSTER, BOBBY W, WICHITA
WEDDLE, DOUGLAS P, FORT SCOTT
WEDEL, KENNETH O, MINNEAPOLIS
WEDEL, KERMIT G, MINNEAPOLIS
WEIDENSAUL, D N, HUTCHINSON
WEIGAND, JOEL T, WELLINGTON
WEIGEL, JOHN W, KANSAS CITY
WEIPPERT, EDWARD J, WICHITA
WEIR, WILLIAM C, ERIE
WELCH, ANNA L, WICHITA
WELCH, JACK W, HALSTEAD
WELCH, LAUREN A, WAMEGO
WELCH, LAUREN K, WICHITA
WELCH, MARTIN H, WICHITA
WELCH, MAURA S, GARDEN CITY
WELL, MICHAEL A, LAWRENCE
WELLER, ELIZABETH B, KANSAS CITY
WELLER, RONALD ALAN, KANSAS CITY
WELLS, ALVIN Y, WINFIELD
WELLS, BRUCE W, WINFIELD
WELLS, MAX MICHAEL, WICHITA
WELLSHEAR, CHARLES C, WICHITA
*WELSH, NANCY JANE, TOPEKA
WELTMER, ROGER P, BELDIT
WENGER, GREGG D, SABETHA
WENINGER, JOHN H, WICHITA
WERNER, WILLARD F, TRIBUNE
WERTH, CLAUDE J, SHAWNEE MISSION
WERTH, DARRELL DEAN, HAYS
WERTZBERGER, JOHN, LAWRENCE
WERTZBERGER, KENNETH LYNN, LAWRENCE
WESBROOK, CLYDE W, BELLEVILLE,IL
WESCOE, W CLARKE, NEW YORK,NY
WEST, WILLIAM T, WICHITA
WHALLON, JACOB T, WICHITA
WHEELER, DWIGHT E, NEWTON
WHEELER, NICKY RAY, WICHITA
WHEELER, PINCKNEY R, WICHITA
WHITAKER, JAMES A, WICHITA
WHITAKER, MARK A, GARDNER
WHITAKER, REN R, OBERLIN
WHITE, CHARLES L, GREAT BEND
WHITE, CHARLES M, WICHITA
WHITE, DONALD C, COFFEYVILLE
WHITE, FAGAN N, RUSSELL
WHITE, JOHN P, PARSONS
WHITE, R BURNLEY, WINFIELD
WHITE, THADDEUS H, MANHATTAN
WHITE II, BENJAMIN E, EL DORADO
WHITEHEAD, RICHARD E, SHAWNEE MISSION
WHITLEY, DOUGLAS M, SHAWNEE MISSION
WIEGMAN, HUGH ALAN, HAYS
WIEGMANN, THOMAS B, KANSAS CITY,MO
WIENS, J WENDELL, NEWTON
WIENS, PETER K, NESS CITY
WIENS, TIMOTHY K, KANSAS CITY,MO
WIGGINTON, GERALD D, D.O., SHAWNEE MISSION
WIGGLESWORTH, ANNE, WAMEGO
WIGGS, JAMES W, GREAT BEND
WIKSTEN, VERNON C, TOPEKA
WILCHINS, LAWRENCE J, LAS VEGAS,NV
WILCOX, DONALD E, TOPEKA
*WILCOX, ROBERT N, WICHITA
WILCOX JR, HOWARD L, HAYS
WILCOX SR, HOWARD L, LAWRENCE
WILDER, LOWELL W, WICHITA
WILDS, CHARLES E, TOPEKA
WILEY, HORACE M, GARDEN CITY
WILEY, JOHN H, SHAWNEE MISSION
WILKINSON, LARRY K, WICHITA
WILLARD EXEC SE, JUDY, ELLINWOOD
WILLIAMS, CHARLES L, WICHITA
WILLIAMS, EVAN ROBERT, DODGE CITY
WILLIAMS, HOMER J, OSAGE CITY
*WILLIAMS, HOWARD V, WICHITA
WILLIAMS JR, STERLING B, KANSAS CITY
WILLIAMSON, JOHN, HUTCHINSON
WILSON, H RANDOLPH, HALSTEAD
WILSON, MARVIN H, TOPEKA
WILSON, ROBERT B, SHAWNEE MISSION
WILSON, ROBERT L, VALLEY CENTER
WILSON, SLOAN J, SHAWNEE MISSION
WINBLAD, J KENT, WINFIELD
WINBLAD, JAMES N, WINFIELD
WINCHELL, H H FORSYTH, WICHITA
WINER, RICHARD S, TOPEKA
WING, NANCY J, TOPEKA
WINTER JR, CALVERT J, KANSAS CITY
WISDOM, JAY K, WICHITA
WISE III, JOSEPH EDWARD, KANSAS CITY
WISNER JR, HARRY J, WICHITA
WISNER JR, JOHN HENRY, KANSAS CITY
WITTMANN, ALBERT F, WICHITA
WOERNER, DAVID R, SALINA
WOLF, CURTIS V, LYONS
WOLF, KARL T, KANSAS CITY
WOLF, PATRICK G, CONCORDIA
WOLFE, FREDERICK, WICHITA
WOLFF, FREDERICK P, PRATT
WOLKOFF, CDR A STARK, SAN FRANSICO,CA
WOLLMANN, MARTIN, LAWRENCE
WONDER II, DONALD K, KANSAS CITY,MO
WONG, BERT Y, KANSAS CITY
WONG, NORMUND, TOPEKA
WOOD, DOUGLAS H, PITTSBURG
WOOD, EDWARD RUSSELL, TOPEKA
WOOD, FRED M, KANSAS CITY
WOOD, GARY B, WICHITA
WOOD, ROBERT D, WICHITA
WOODHOUSE, CHARLES L, WICHITA
WOODS, HUGH J, SMITH CENTER
WOODS, ROBERT P, TOPEKA
*WORSING JR, ROBERT A, WICHITA
WORTMAN, JACK A, HUTCHINSON
WRAY, ALEXANDER J, WICHITA
WRIGHT, KENDALL M, EMPORIA
WRIGHT, STANLEY E, JUNCTION CITY
WRIGHT III, ROBERT W, KANSAS CITY
WRIGHT JR, ROBERT W, KANSAS CITY
WU, JIN-TZE, WICHITA
WULFF, EDWIN T, ATCHISON
WURM, MICHAEL J, PHILLIPSBURG
WURSTER, GEORGE R, SHAWNEE MISSION

Y

YAGHMOUR, TALAAT E, PITTSBURG
YAKAR, DANIEL, KANSAS CITY
YE, RICHARD C, SHAWNEE MISSION
YEOMANS, RONALD N, OLATHE
*YOCKEY, CHARLES C, WICHITA
YODER, EMERSON D, DENTON
YODER, VERNON E, HESSTON
YOHE, RUTH M, SHAWNEE MISSION
*YOON, CHANG SUP, WICHITA
YORKE JR, CRAIG H, TOPEKA
YOUMANS, ROGER L, AFRICA,
YOUNG, CHARLES H, ATCHISON
YOUNG, DOUGLAS L, WICHITA
YOUNG, JOHN W, SHAWNEE MISSION
YOUNG, PAUL E, TOPEKA
*YOUNG, ROBERT, WICHITA
YOUNG, THEODORE E, TOPEKA
YOUNGBERG, DEAN I, WICHITA
YOUNGLOVE, R HAL, SHAWNEE MISSION
YOUNGSTROM, KARL A, SHAWNEE MISSION
YULICH, JOHN O, SABETHA

Z

ZABEL, KENNETH P, PITTSBURG
ZACHARIAS, CARL KURT, DODGE CITY
ZACHARIAS, DAVID LLOYD, TOPEKA
ZACK, ASHLEY S, SHAWNEE MISSION
ZAINALI, ASSADOLLAH, LIBERAL
ZAMIEROWSKI, DAVID S, SHAWNEE MISSION
ZAREMSKI, SHERMAN C, KANSAS CITY
ZARNOW, HILARY, WICHITA
ZATZKIN, JAY B, WICHITA
ZELLER, MYRON J, GARDEN CITY
*ZEPICK, LYLE F, WICHITA
ZIEGLER, DEWEY K, KANSAS CITY
ZIMMERMAN, DANIEL D, SHAWNEE MISSION
ZIMMERMAN, KENNETH D, WICHITA
ZIMMERMAN, WILLIAM H, TOPEKA
ZINN, THOMAS W, KANSAS CITY
ZONGKER, PHILIP E, WICHITA

* Probationary members.

Medical Specialty Codes Used in This Directory

A	Allergy
ADM	Administrative Medicine
ADT	Addictionology
AM	Aviation Medicine
ANES	Anesthesiology
CD	Cardiovascular Disease
CDS	Cardiovascular Surgery
CDTS	Cardiovascular & Thoracic Surgery
CHN	Child Neurology
CHP	Child Psychiatry
CP	Clinical Pharmacology
D	Dermatology
DR	Diagnostic Roentgenology
EENT	Eye, Ear, Nose and Throat
EM	Emergency Medicine
END	Endocrinology
ENT	Ear, Nose, Throat
ES	Endoscopy Surgery
FM	Family Medicine
FP	Family Practice
GE	Gastroenterology
GEN	Genetics
GER	Geriatrics
GP	General Practice
GPM	General Preventive Medicine
GPVS	General & Peripheral Vascular Surgery
GS	General Surgery
GYN	Gynecology
HEM	Hematology
ID	Infectious Diseases
IE	Insurance Examination
IM	Internal Medicine
MDST	Medical Student
MFM	Maternal Fetal Medicine
MO	Medical Oncology
N	Neurology
NEP	Nephrology
NM	Nuclear Medicine
NEO	Neonatology
NP	Neuropsychiatry
NR	Nuclear Radiology
NS	Neurological Surgery
OBG	Obstetrics and Gynecolog
OM	Occupational Medicine
ON	Oncology
OPH	Ophthalmology
ORS	Orthopedic Surgery
OS	Other Specialties
OST	Osteopathy
OTO	Otorhinolaryngology
P	Psychiatry
PATH	Pathology
PD	Pediatrics
PDA	Pediatric Allergy
PDC	Pediatric Cardiology
PDN	Pediatric Neurology
PDO	Pediatric Ophthalmology
PDR	Pediatric Radiology
PDS	Pediatric Surgery
PH	Public Health
PM	Physical Medicine & Rehabilitation
PNP	Pediatric Nephrology
PS	Plastic Surgery
PUD	Pulmonary Disease
R	Radiology
RES	Resident
RHU	Rheumatology
RM	Rehabilitative Medicine
RN	Radiology & Neurology
RO	Radiology/Oncology
RT	Radiation Therapy
SM	Sports Medicine
SON	Surgical Oncology
TR	Therapeutic Radiology
TS	Thoracic Surgery
U	Urology
00	Retired

Physician Distribution by Cities

EXPLANATION OF CODES USED IN THIS SECTION

Line 1: Doe, John R., 1234 Oak St., 67052
(Name) (Street Address) (Zip Code)

Line 2: (654-2222)
(Telephone Number)

Line 3: 33 M 1902 58 FP
(Year of Birth) (Sex) (Medical School) (Year of Graduation) (Specialty)

Telephone area code follows city name. * Probationary Members

ABILENE—913
(Dickinson County Society)

BERKLEY, DON H, 1111 N BRADY,67410
263-4131
35 M 1902 61 FP
BIGGS, DENNIS, 1405 N CEDAR,67410
263-7190
48 M 1902 74 FP
CHAFFEE, DEAN C, RR 1,67410
263-4131
11 M 1902 44 FP
COLEMAN, GARY, 1405 N CEDAR,67410
263-7190
46 M 1902 72 FP
MOHLER, JACK M, 515 NE TENTH,67410
263-1419
32 M 1902 61 PM
NARCISO, VICENTE D, 515 NE 10TH ST,67410
263-2253
45 M 74810 68 GS
RORABAUGH, DONALD C, PROFESSIONAL BLDG,67410
263-4131
33 M 1902 58 FP
SCHWARTING, J STEVE, 1405 N CEDAR,67410
263-7190
46 M 3401 72 FP
SHEERN, MARK DOUGLAS, 1111 N BRADY,67410
263-4131
51 M 1902 76 FP
SONGER, HERBERT L, 811 SPRUCE WAY,67410
\-
12 M 1902 38 OO

ALMA—913
(Pottawatomie County Society)

MCKNIGHT, ELLIS B, ,66401
765-3317
03 M 1902 33 FP

ALTAMONT—316
(Labette County Society)

JACKSON, VICTOR L, BOX 467,67330
784-5318
20 M 2105 50 FP

ANDALE—316
(Sedgwick County Society)

GOODWIN, MARY K, ANDALE CLINIC,67001
267-0865
53 F 1902 77 FP
STECH, JOSEPH M, ANDALE CLINIC,67001
445-2152
27 M 3006 56 FP

ANDOVER—316
(Sedgwick County Society)

ESTRADA, EDMUNDO C, 327 W CENTRAL,67002
733-1331
43 M 74801 67 GS
ESTRADA, LINA, 327 W CENTRAL,67002
733-1331
43 F 74801 68 PD

ANTHONY—316
(Tri-County Society)

ANTRIM, PHILIP JENIFER, 1101 E SPRING,67003
842-5144
15 M 1902 42 FP
HALL, JERRY D, 1101 E SPRING,67003
842-5144
31 M 3901 56 FP

ARKANSAS CITY—316
(Cowley County Society)

ALVAREZ, NORBERTO, 112 E CENTRAL,67005
442-4850
27 M 27501 53 FP
AUCAR, ALFREDO, BOX 1105,67005
442-1710
23 M 27501 53 OTO
BAKER DO, ROBERT R, 2508 EDGEMONT BOX 714,67005
442-8924
50 M 2878 78 FP
CAMPBELL, GARLAND L, 114 W WALNUT,67005
442-1350
13 M 1902 40 U
GREEN, LAWRENCE C, PO BOX 173,67005
442-4850
43 M 3901 69 FP
HERNANDEZ, MANUEL, 515 N SUMMIT,67005
442-4850
45 M 84701 69 GS
HINSHAW, EDGAR D, RT #3,67005
442-1273
15 M 1902 51 R
LEIKER, JOSEPH, 112 E CENTRAL,67005
442-7900
48 M 1902 73 IM
OLD, JERRY L, PO BOX 1148,67005
442-2100
49 M 1902 74 FP
PEREIRA, WILLY G, 2508 EDGEMONT DR,67005
442-8540
39 M 73701 67 IM
ROSS, DAVID K, PO BOX 1148,67005
442-2100
48 M 1902 75 FP
SINGH, GIRVAR, 2508 EDGEMONT DR,67005
442-4300
40 M 49555 64 OPH
SMITH, BRUCE G, 115 E RADIO LANE,67005
442-5600
20 M 1902 44 IM
SMITH, NEWTON C, PO BOX 1148,67005
442-2100
21 M 3901 45 FP

ARMA—316
(Crawford County Society)

RIGLER, WILSON F, 511 WASHINGTON,66712
347-8619
42 M 1803 69 FP

ATCHISON—913
(Atchison County Society)

BOSSE, FRANK K, 1301 RIVERVIEW DRIVE,66002
\-
09 M 2802 33 OO

BRADY, CHARLES S, 111 N 5TH ST,66002
367-1232
11 M 3006 38 GS
BRIBACH, EUGENE J, 125 1/2 N 5TH,66002
367-0225
83 M 2802 05 OPH
BROWN, ROBERT O, 1400 NORTH 2ND,66002
367-1922
14 M 1902 44 FP
BURKE, JOSEPH V, 1301 N 3RD,66002
367-5496
35 M 3006 66 GS
FAST, ROBERT E, 1225 N 2ND,66002
367-0362
48 M 1902 74 OBG
FAST, W SPENCER, RAMSAY MED CLINIC,66002
367-0362
11 M 3006 39 FP
GROWNEY, JOHN T, 801 ATCHISON,66002
367-5020
37 M 3006 63 FP
HART, LAWRENCE E, 1412 N 2ND,66002
367-5054
32 M 1902 64 FP
MORRISON, IRA R, 825 N 10TH,66002
-
07 M 1611 36 IM
RIDER, JAMES W, 1225 N 2ND ST,66002
367-0362
47 M 2803 73 FP
TAYIEM, ABDEL K, 1225 N 2ND,66002
367-0362
43 M 33002 68 GS
TIVORSAK, ARKOM, 1716 COUNTRY LANE,66002
367-2131
40 M 89101 68 R
WALLACE JR, WAYNE O, 1301 N THIRD,66002
367-7300
36 M 2803 65 FP
WULFF, EDWIN T, 923 N FIFTH,66002
367-5033
07 M 2834 36 FP
YOUNG, CHARLES H, 1301 N 3RD,66002
367-4053
23 M 1902 53 FP

ATTICA—316
(Tri-County Society)

STONE, GRANT C, 500 N HARPER,67009
254-7219
08 M 5605 35 FP

ATWOOD—316
(Northwest Kansas Society)

DILL, RODNEY, PO BOX 67,67730
626-3229
41 M 74811 77 GP

AUGUSTA—316
(Butler-Greenwood Society)

ANDERSON, DALE W, 209 D WEST 7TH STREET,67010
775-5432
30 M 1902 55 FP
BARBER, JAMES L, AUGUSTA PLAZA,67010
775-5432
31 M 1902 57 FP
TUONG, TRAN MANH, AUGUSTA PLAZA,67010
775-5432
39 M 94101 65 GP

BAXTER SPRINGS—316
(Cherokee County Society)

ALQUIST, VERYL D, 21ST & FAIRVIEW,66713
623-4942
17 M 1902 42 GS
CHUBB, RICHARD M, 445 EAST 10TH,66713
856-2144
29 M 1506 54 FP
FITZGERALD, THOMAS J, 445 E TENTH - BOX 578,66713
856-2144
45 M 74807 79 GP
MEHAFFY, ORVILLE A, 411 E 11TH,66713
856-2383
42 M 1902 69 FP

BELLEVILLE—913
(Republic County Society)

BEIDERWELL, PAUL L, 2703 M ST,66935
-
08 M 3901 38 FP
CHANEY, ERNIE J, 2316 G STREET,66935
527-2237
27 M 1902 56 FP
DOUBEK, HERBERT D, PO BOX 250,66935
527-2237
28 M 1902 56 FP
SCOTT, DUANE L, BELLEVILLE CLINIC,66935
527-2217
34 M 1902 60 FP
WARD, JAMES A, 1206 18TH ST,66935
527-2217
34 M 1902 58 FP

BELOIT—913
(Mitchell County Society)

DOBRATZ, ROBERT A, 310 W 8TH,67420
738-2246
24 M 1902 52 FP
DRAKE, DOUGLAS J, MEDICAL CENTER,67420
738-2246
43 M 1902 71 FP
KLENDA JR, MARTIN B, BELOIT MED CENTER,67420
738-2245
38 M 1643 63 GS
LONEY, JOHN M, 310 WEST 8TH,67420
738-2246
50 M 1902 74 IM
WELTMER, ROGER P, 112 W MAIN,67420
738-2574
18 M 1902 44 U

BLUE RAPIDS—913
(Northeast Kansas Society)

LAWLESS, HAROLD L, 607 LINCOLN,66411
226-7202
29 M 702 54 FP

BONNER SPRINGS—913
(Wyandotte County Society)

MAY, KENNETH L, 122 N NETTLETON,66012
422-2020
20 M 1902 51 FP
MITTS, ERNEST W, 122 N NETTLETON,66012
422-2020
22 M 1902 51 FP
WAGGONER, FRANKLIN E, 122 N NETTLETON,66012
422-2020
26 M 1902 61 FP

BUHLER—316
(Reno County Society)

FRIESEN, ORLANDO J, 107 W 2ND,67522
543-2330
27 M 1902 56 FP

BURLINGAME—913
(Shawnee County Society)

GOERING, EMIL L, 130 W SANTA FE,66413
654-2300
27 M 1902 57 IM

CALDWELL—316
(Tri-County Society)

KINNAN, L F, 523 S MARKET,67022
845-6422
18 M 3901 42 FP

CANEY—316
(Southeast Kansas Society)

MOORE, ROBERT F, 4TH & MCGEE,67333
879-213S
28 M 1902 56 FP

CEDAR VALE—316
(Southeast Kansas Society

COHN8ERG, ROSELLEN E, PO BOX 398,67024
7S8-2266
22 F 2802 47 FP

(CHANUTE—316
(Southeast Kansas Society)

ABBUEHL, DON R, 505 SOUTH PLUMMER,66720
431-2S00
18 M 1902 44 GS
ASHLEY, SAMUEL G, 505 SOUTH PLUMMER,66720
431-2500
16 M 1902 43 FP
BAKER, HENRY K, 811 WEST MAIN,66720
431-1600
08 M 1606 35 GS
BURKMAN, REUBEN J, 1S01 W 7TH,66720
431-9310
28 M 1902 54 FP
DICK JR, HENRY J, 1S01 W 7TH,66720
431-9310
27 M 1902 58 FP
GEHRT, EARL B, S05 SO PLUMMER,66720
431-2S00
32 M 1902 62 FP
HASKINS, ROBERT J, 505 S PLUMMER,66720
431-2S00
46 M 1902 74 FP
KIHM, ALBERT A, 50S S PLUMMER,66720
431-2S00
27 M 1902 5S FP
MIH, ALEXANDER, 1002 WEST 4TH,66720
473-2227
22 M 24209 47 ANES
PARHAM, VERDON W, 505 S PLUMMER,66720
431-2S00
47 M 1902 73 FP
VAN HOUDEN, CHARLES E, 505 S PLUMMER,66720
431-2S00
S2 M 1902 77 GS

CHAPMAN—316
(Dickinson County Society)

SVOBODA, CHARLES R, 413 N MARSHALL,67431
922-6400
18 M 1902 46 FP

CHETOPA—316
(Labette County Society)

PEFFLY, ELMER D, 327 MAPLE,67336
236-7188
22 M 3901 53 FP

CLAY CENTER—913
(Clay County Society)

ANDERSON, SEVERT A, 1749 8ERGLJND DR,67432
-
07 M 1902 36 OD
CARLETON, RICHARD C, 709 LIBERTY,67432
632-5603
31 M 3005 61 FP
DALUM, PETER JOSEPH, 709 LIBERTY SUITE C,67432
632-2181
45 M 2803 76 FP
GOOD, MICHAEL W, 709 LIBERTY,67432
632-2181
S3 M 1902 78 FP
MCVAY, R 8RUCE, 1503 FIFTH ST,67432
-
97 M 1902 29 OD
O'DONNELL, RICHARD H, 709 LIBERTY,67432
632-3101
16 M 1902 41 GS
RICHARDS, DENNIS D, 115 S 6TH,67432
632-5621
34 M 1902 60 FP
STEIN8AUER, JEFFREY ROBERT, 709 LIBERTY,67432
632-2181
51 M 1902 77 FP

CLYDE—913
(Cloud County Society)

FREEBORN JR, WARREN S,66938
446-2221
26 M 1720 51 FP

COFFEYVILLE—316
(Southeast Kansas Society)

8ANKS, GILBERT, COFFEYVILLE DOCTORS' CL,67337
251-7S00
49 M 2401 75 1M
CAMP8ELL, WILLIAM H, 1411 W 4TH,67337
251-323S
39 M 1902 65 OPH
COYLE, JOHN F, PO BOX 487,67337
251-2400
21 M 1902 44 FP
DICKINSON, CHARLES R, 108 W 7TH,67337
251-1340
20 M 1606 44 GS
DIXON, RAYMOND W, 1411 W 4TH,67337
251-1090
46 M 4706 71 GS
EDROZO, M LUZ LUNA, PO BOX 856,67337
2S1-1200
F 74801 68 PATH
ELLIS, STEPHEN S, 1411 W 4TH,67337
251-3360
11 M 2802 36 GS
GIBBS, EUGENE, PO BOX 716,67337
251-7260
M 64914 68 FP
HA, SANG W, S04 WILSHIRE,67337
2S1-77S0
35 M 58309 60 O8G
HAN, CHAN S, 908 SIGGINS,67337
251-1S60
35 M S8306 61 PD
HOFFMAN, CARL, 1411 W FOURTH STE D,67337
2S1-3235
OPH
HOWERTER JR, BERNARD E, PO 80X 659,67337
251-4790
43 M 1803 68 U
JIRICKO, MILOS, 2510 W EIGHTH,67337
2S1-1200
37 M 28501 61 ANES
NICHOLS, RICHARD, 1411 W FOURTH,67337
2S1-6540
M 1803 73 ORS
READ, WILLIAM T, 1411 WEST 4TH,67337
251-1120
16 M 2802 40 FP
SANDHU, PAUL S, PO BOX 257,67337
2S1-24S0
42 M 49S08 6S GS
UY, WILSON O, COFFEYVILLE MEM HOSPITAL,67337
2S1-1200
42 M 74801 67 PATH
VAKAS, JOHN L, 1S08 W 4TH,67337
2S1-3443
38 M 1902 64 1M
WHITE, DONALD C, PO BOX 262,67337
251-1200
35 M 3S1S 6S R

COLBY—913
(Northwest Kansas Society)

DAHL, ASHER W, COLBY CLINIC,67701
462-3333
28 M 1902 S8 FP
HASSETT, GERARD R, 187S HARVEY,67701
-
24 M 3006 S0 R

HILDYARD II, VICTOR H, BOX 28,67701
462-3332
47 M 702 73 FP
JACOBSEN, DWIGHT SKINNER, COLBY CLINIC,67701
462-3333
31 M 3S45 60 GS
MARSHALL, GEORGE D, COLBY CLINIC,67701
462-3333
09 M 1902 36 FP
REGIER, LADONNA M, COLBY CLINIC,67701
462-3332
47 F 1902 73 FP
SMITH JR, FLOYD L, COLBY CLINIC,67701
462-3333
20 M 1902 44 FP

COLDWATER—316
(Iroquois County Society)

GOERING, DONALD O, BOX 748,67029
582-2136
31 M 1902 S6 FP

COLUMBUS—316
(Cherokee County Society)

BELCHER, GEORGE O, BOX 309,66725
429-25S7
34 M 1902 60 FP
JONES, FORREST H, 219 S KANSAS,66725
429-3744
25 M 1902 54 FP
PASIMIO, ROGER S, R2 BOX 259,66725
429-1977
38 M 74801 62 GS

COLWICH—316
(Sedgwick County Society)

LIES, BARTHEL N, 309 S THIRD,67030
—
11 M 2802 37 OO

CONCORDIA—913
(Cloud County Society)

BRAY, AVIS PAGE, 1010 3RD AVE,66901
243-1S60
17 F 702 54 FP
BUTT, MUHAMMAD, CONCORDIA MED GROUP,66901
243-1S60
45 M 70401 69 GS
COULTER OD, THAYNE A, 11TH & WASHINGTON,66901
243-4455
12 M 2878 37 GP
FOWLER, WAYNE L, CONCORDIA MED GROUP,66901
423-1560
23 M 1720 47 IM
GELVIN, E RAYMOND, 835 WEST 9TH,66901
243-1S60
03 M 3005 27 GS
HOFER, DEWAYNE O, COUNTRY CLUB DR,66901
243-1263
36 M 1606 62 R
KOSAR, CLARENCE O, BOX 362,66901
—
98 M 1902 26 OO
LAWTON, MARVIN K, CONCORDIA MED GROUP,66901
243-1S60
31 M 3005 58 GS
LLOYD, JAMES W, 910 WEST 11TH,66901
243-7011
44 M 1902 73 FP
MOORE, JAMES E, PO BOX 305,66901
243-1094
48 M 1902 74 P
NELSON, PAUL L, 1010 THIRD AVE,66901
243-1560
27 M 1902 55 PD
NEWMAN, CARL T, PO BOX 587,66901
243-2S11
49 M 64914 76 U
NIXON, RICHARD R, 519 WASHINGTON,66901
243-1263
32 M 1643 57 R
OWENSBY, L C, 222 W SIXTH,66901
243-3386
21 M 2902 44 OPH

RUZICKA, LAWRENCE J, 1010 3RD AVE,66901
243-1560
13 M 300S 40 ANES
SAUNDERS, MICHAEL E, 1404 CRESCENT LANE,66901
243-1S60
50 M 1902 73 IM
STRYKER JR, HENRY B, 717 FIRST AVE,66901
—
19 M 3S01 44 OO
THORNTON JR, FOXHALL P, 1010 3RD AVE,66901
243-1S60
25 M 5101 50 IM
WOLF, PATRICK G, 1413 COUNTRY CLUB DR, 66901
—
52 M

COTTONWOOD FALLS—316
(Flint Hills Society)

BROWNING, JIMMIE L, 3243 E MURDOCK,66845
688-3070
SO M 1902 78 FP
MCKEE, LEO F, ,66845
273-6681
16 M 1902 39 FP

COUNCIL GROVE—316
(Flint Hills Society)

BARKER, ROYAL A, 221 HOCKADAY,66846
767-5126
21 M 1902 53 FP
BLACKBURN, ROBERT W, 221 HOCKADAY,66846
767-S125
22 M 1902 49 FP
SCHULTZ, JAMES E, 221 HOCKADAY,66846
767-S125
26 M 1902 56 FP

CUNNINGHAM—316
(Wyandotte Society)

ALLBRITTEN JR, FRANK F, PO BOX 177,67035
—
14 M 4101 38 OO

DE SOTO—913
(Johnson County Society)

POMMERENKE, FORREST A, 415 E SECOND,66018
585-1832
48 M 1902 75 FP

DENTON—913
(Northeast Kansas Society)

YODER, EMERSON O, ,66017
3S9-6S31
14 M 1902 49 FP

DERBY—316
(Sedgwick County Society)

MCKERRACHER, ROBERT O, 400 A NORTH BALTIMORE,67037
688-1779
27 M 3901 55 FP
MILLER, LAWRENCE H, 1410 N WOODLAWN,67037
788-3741
40 M 1001 67 FP
MOYER, HERMAN J, 200 S BALTIMORE,67037
788-1484
24 M 3901 55 FP
VINZANT, MARK N, 1410 N WOODLAWN,67037
681-3092
45 M 64914 75 FP

DIGHTON—316
(Southwest Kansas Society)

VON LEONROD JR, GEORGE, PO BOX 146,67839
397-5314
16 M 1902 43 FP

DODGE CITY—316
(Ford County Society)

AMAWI, MOHAMMAD S, DODGE CITY MED CENTER,67801
225-1371
46 M 87501 71 GS
AVILA, OSCAR A, DODGE CITY MED CENTER,67801
225-1371
41 M 17603 69 IM
AYUTHIA, ISSARA I, 2004 FREDERICK DR,67801
-
40 M 89101 66 PATH
BAUM, ARNOLD H, DODGE CITY MED CENTER,67801
225-1371
16 M 2101 44 OBG
BETTERTON, DALE C, PO BOX 1413,67801
225-0200
38 M 2604 73 FP
BOLES, R DALE, DODGE CITY MED CENTER,67801
225-1371
23 M 3901 53 PD
BROWNRIGG, RICHARD L, 108 ROSS BLVD,67801
225-0075
35 M 1902 61 U
BUSCH, ANTHONY B, SOUTHWEST CLINIC,67801
-
05 M 2002 31 DO
CHOTIMONGKOL, ANUPONG, DODGE CITY MED CENTER,67801
225-1371
43 M 89102 69 OBG
CONARD, CLAIR C, DODGE CITY MED CENTER,67801
225-1371
27 M 1902 55 IM
GARCIA, GUILLERMO D, 2020 CENTRAL,67801
225-1371
43 M 23101 68 ORS
GREENBERG, GEORGE E, 1904 BURR PARKWAY,67801
276-8241
42 M 401 68 R
JOHNSON, HOWELL D, DODGE CITY MED CENTER,67801
225-1371
45 M 1902 71 IM
MCCOY, RONALD, SOUTHWEST CLINIC,67801
227-3141
19 M 3901 47 FP
MCELHINNEY, CHARLES F, DODGE CITY MED CENTER,67801
227-1371
36 M 1902 62 GS
MCGINNIS, MICHAEL J, SOUTHWEST CLINIC,67801
227-3141
41 M S501 69 GS
MIMOSO, JOSE J, SOUTHWEST CLINIC,67801
227-3141
37 M 4201 61 OBG
OHMAN, RICHARD J, 1810 1/2 FAIRWAY DR,67801
-
15 M 2407 41 DO
REDDY, SATTI S, 108 RUSS BLVD,67801
225-0075
35 M 49504 66 U
SCHWARTZ, EUGENE W, 1ST NATL BANK BLDG,67801
225-4261
24 M 1902 50 OPH
STOCKWELL, MORGAN D, 2020 CENTRAL,67801
225-1371
24 M 1902 55 IM
TREKELL, WILLIAM V, SOUTHWEST CLINIC,67801
227-3141
25 M 1902 52 ORS
TROTTER, ROGER COURTNEY, 1111 SIXTH AVE,67801
225-6120
47 M 1902 74 FP
VIERTHALER, CARL A, DODGE CITY MED CENTER,67801
225-1371
53 M 1902 78 IM
WILLIAMS, EVAN ROBERT, DODGE CITY MED CENTER,67801
225-1371
25 M 1606 52 GS
ZACHARIAS, CARL KURT, DODGE CITY MED CENTER,67801
225-1371
21 M 40707 47 ORS

EDWARDSVILLE—913
(Wyandotte County Society)

SOUTHWICK, CHIRSTOPHER L, 441 BEACH,66111
-
56 M 1902

EL DORADO—316
(Butler-Greenwood Society)

BRIAN, ROBERT M, 123 N ATCHISON,67042
321-1230
02 M 1606 30 FP
DELLETT, KENNETH B, 3RD & VINE,67042
321-1910
30 M 1902 55 OPH
GIROD, CHARLES I, 123 N ATCHISON ST #103,67042
321-4981
11 M 4706 44 GS
HAFFNER, WILLIAM N, 123 N ATCHISON,67042
321-5049
35 M 1902 61 GS
JACOB, KANNAMPALLY L, 123 N ATCHISON,67042
321-0056
31 M 49537 59 U
KASSEBAUM, GLEN E, 123 N ATCHISON,67042
-
98 M 1606 23 GS
LEE, YONG U, 123 N ATCHISON,67042
321-5630
35 M 59310 60 GS
OLSEN, PHILLIP S, 123 N ATCHISON,67042
321-2100
46 M 1902 73 IM
OVERHOLSER, NORMAN H, 119 N JONES,67042
321-2010
16 M 1902 41 FP
PROCTOR, ROBERT W, 123 N JONES,67042
321-2010
38 M 1902 63 FP
REDDY, VENUMBAKA C, ROOM 302,67042
321-3300
46 M 49511 70 IM
SANDBERG, CHRIS B, 119 NORTH JONES,67042
321-2010
48 M 1902 74 FP
SHIELDS JR, JAMES M, 119 N JONES,67042
321-2010
18 M 4802 42 FP
SIWEK, CHRISTOPHER W, 123 N ATCHISON,67042
321-5211
48 M 75911 71 ORS
WHITE II, BENJAMIN E, 119 N JONES,67042
321-2010
27 M 1902 54 FP

ELKHART—316
(Seward County Society)

CABRERA, ALBERTO, BOX 997,67950
697-2155
30 M 74901 55 GS
IWAY, BELINO D, 411 SUNSET BOX 460,67950
697-2175
42 M 74811 66 IM
IWAY, OLIVIA N, BOX 460 - 411 SUNSET,67950
697-2175
43 F 74811 68 PD
PERIDO, DOMINADOR T, BOX 997,67950
697-2155
44 M 74901 68 GS

ELLINWOOD—316
(Barton County Society)

LAW, FINDLEY, MEDICAL ARTS BLDG,67526
564-2170
22 M 1902 51 FP
*WILLARD EXEC SEC, JUDY, BARTON COUNTY MED SOCIETY,67526
792-4391

ELLIS—913
(Central Kansas Society)

ROCHANAYON, PIRA, 1204 WASHINGTON,67637
726-4956
43 M 89101 69 FP
SURFACE, GARDNER A, 101 WEST 13TH,67637
726-4454
02 M 1902 29 DO

ELLSWORTH—913
(Central Kansas Society)

DAVIS, GEORGE R, 308 KINGSLEY,67439
472-3121
19 M 1902 44 GS
O'DONNELL, HAROLD F, 412 BLAKE,67439
-
99 M 1902 26 DO

SEITZ JR, JOSEPH E. 308 KINGSLEY,67439
472-3121
22 M 1902 46 FP

EMPORIA—316
(Flint Hills Society)

AMENO, OOUGLAS J. 827 COMMERCIAL,66801
343-6565
46 M 1902 79 OBG
8EST, JOHN F. 919 W 12TH,66801
342-2521
51 M 2803 77 IM
8OSILJEVAC JR, JOSEPH E. 100S W 12TH,66801
343-2336
51 M 1902 75 TS
8RADLEY, H RUSSELL, 1601 STATE,66801
343-2900
35 M 1902 61 FP
8ROCKHOUSE, JOHN P. 1601 STATE,66801
343-2900
31 M 1902 57 IM
8URGESON, FRANK G. 1601 STATE,66801
342-6989
40 M 300S 65 OPH
8UTCHER, THOMAS P. 1128 LAWRENCE,66801
342-0722
05 M 1601 34 GS
CAMP8ELL, EOWARD G. 1601 STATE,66801
343-2900
31 M 1902 61 FP
COLOSMITH, OONALO C. 1024 W 12TH,66801
342-7047
26 M 1902 58 FP
OAVIS, OAVIO R. 102S STATE ST APT #2,66801
-
02 M 2101 28 OO
EOWAROS, OAVIO J. 1601 STATE ST,66801
343-1191
43 M 2803 69 ORS
ELLIS, 80B8Y J. 1601 STATE,66801
343-2900
51 M 1902 77 IM
FRAZIER, RICHARO L. 1005 W 12TH,66801
342-5137
40 M 1902 72 GS
GANN, E LAMONTE, RR #2,66801
-
07 M 2802 37 OO
GARCIA, GOULO C. 919 WEST 12TH AVE,66801
342-2521
32 M 3607 58 IM
GEITZ, JAMES M. 919 W 12TH,66801
342-2521
46 M 1902 72 IM
GINAVAN, OUANE A. 1024 W 12TH,66801
342-5876
35 M 1902 62 FP
GLENN, JAMES N. 1601 STATE,66801
343-1191
40 M 4804 66 ORS
HARVEY, JOHN E. 2506 W 15TH,66801
343-2900
39 M 1902 65 OBG
HAVENHILL II, MARSHALL A. 812 ANOERSON ST,66801
343-6400
35 M 1902 61 OBG
HOPPER, CHARLES R. 25 W 5TH,66801
342-2341
17 M 1902 47 FP
HOWELL, BARBARA JOYCE, 2510 W 15TH,66801
343-7590
45 F 3401 78 PO
KEITH, ROBERT MARSHALL, 705 S COMMERCIAL,66801
342-0548
27 M 801 54 P
KELLING, COLLYER, 1614 E WILMAN,66801
343-7676
50 M 1902 75 R
KNECHT, STEPHEN M. NEWMAN MEMORIAL HOSP,66801
342-7722
44 M 1902 70 R
KRETSINGER OO, W BROCK, 1601 STATE,66801
343-2393
48 M 2878 77 IM
LOHMEYER, KENNETH L. 1024 WEST 12TH,66801
342-5622
18 M 1902 44 FP
LUEOTKE, WALTER E. NEWMAN MEM HOS,66801
343-6800
18 M 5605 43 PATH
MIGUELINO, OLIVER M. NEWMAN HOSP,66801
343-6800
35 M 74901 57 PATH
MORGAN, JOHN L. 919 WEST 12TH,66801
342-2521
15 M 4101 40 IM
NEIGHBOR, RALPH M. 827 COMMERCIAL ST,66801
343-6565
45 M 1902 72 OBG
NEUER, FREOERICK S. NEWMAN HOSP,66801
342-7722
46 M 3601 71 R
PARKER, WAYNE G. EMPORIA STATE UNIVERSITY,66801
343-1200
27 M 1902 56 FP
PASTOR, VICTOR HUGO, 1601 STATE,66801
342-7715
43 M 13202 68 U
RYAN, W SCOTT, 2510 W FIFTEENTH,66801
343-7590
47 M 1902 73 PO
SAUL, F WILLIAM, ROUTE #5,66801
-
07 M 4113 40 OO
SCHELLINGER, RICHARO P. 1128 LAWRENCE,66801
342-0722
22 M 3005 49 GS
SNOWBARGER, MARVIN O. 1601 STATE STREET,66801
343-2900
29 M 1902 55 FP
SPEARS, CHESTER A. 12TH & CHESTNUT,66801
343-6800
50 M 2834 76 PATH
TRIMBLE SR, OAVIO P. 517 MERCHANT STREET,66801
342-2572
04 M 1902 32 OPH
UNOERWOOO, CHARLES C. 25 WEST 5TH,66801
342-2341
07 M 1902 32 IM
VANOER VELOE, STANLEY L. 1601 STATE,66801
343-2900
16 M 1902 43 GS
WRIGHT, KENOALL M. 1024 WEST 12TH,66801
343-2376
45 M 1902 71 FP

ERIE—316
(Southeast Kansas Society)

BRYAN, EMERY C. RT 2 BOX 171,66733
-
04 M 1902 32 OO
WEIR, WILLIAM C. 115 S MAIN,66733
-
17 M 2501 42 OO

ESKRIDGE—913
(Flint Hills Society)

WALKER, WILLIAM H. 2ND & CEOAR,66423
-
10 M 2401 38 IM

EUDORA—913
(Douglas County Society)

HOLLAOAY, KENNETH R. 101 WEST 10TH,66025
542-2345
34 M 1902 58 FP

EUREKA—316
(Butler-Greenwood Society)

CISKEY, WILLIAM J. PO BOX 310,67045
583-7401
47 M 1902 72 FP
SKAER, STANLEY ALLEN, 100 E 16TH,67045
583-7486
40 M 3901 65 GS

FORT SCOTT—316
(Bourbon County Society)

AKERS, GUY I. 710 W 8TH,66701
223-3100
20 M 1902 53 FP

ALOIS, HENRY, 710 W 8TH,66701
223-3100
13 M 1902 41 OBG
ALOIS, WILLIAM, 710 WEST 8TH,66701
223-3100
20 M 1902 44 GS
BASHAM, JAMES J, 702 MEADOW LANE,66701
-
14 M 1902 37 OO
BENAGE, JOHN F, 821 BURKE,66701
223-2200
32 M 1902 58 OBG
BRAUN, EDWARD W, 710 WEST 8TH,66701
223-3100
42 M 1902 68 U
BURKE, JAMES J, 710 W 8TH,66701
223-3100
35 M 2834 61 IM
CHOW, STANLEY Y, 821 BURKE,66701
223-2200
18 M 24222 39 R
DUNSHEE, CARLYLE M, 710 W 8TH,66701
223-3100
32 M 1902 57 GS
GETTLER, DEAN T, 710 WEST 8TH,66701
223-3100
31 M 1902 57 GS
GOOD, JAMES T, 821 BURKE,66701
223-2200
21 M 2802 45 PATH
GRIMALDI, GARY A, 710 W EIGHTH,66701
223-3100
49 M 1902 74 OBG
IRBY, ADDISON C, 416 S JUDSON,66701
-
05 M 1606 28 OO
IRBY, PRATT, 710 WEST 8TH,66701
223-3100
13 M 4705 36 U
MCCANN, PATRICK E, 710 WEST 8TH,66701
223-3100
28 M 1902 59 IM
MCKENNA, MICHAEL J, 323 S JUDSON SUITE 120,66701
223-3950
38 M 1902 64 FP
NELSON, T EUGENE, 710 W 8TH,66701
223-3100
41 M 1902 69 FP
NICHOLS, ROBERT R, 102 S JUDSON,66701
223-4100
50 M 2803 76 FM
PHELPS, DAVID WAYNE, 102 S JUDSON,66701
223-4100
51 M 1902 76 FP
REEVES, CHARLES S, 710 W 8TH,66701
223-3100
37 M 1902 63 IM
SPENCER, JOHN HAROLD, 710 W 8TH,66701
223-3100
47 M 1902 74 FP
WEDDLE, DOUGLAS P, 710 WEST 8TH,66701
223-3100
43 M 1720 69 FP

FREDONIA—316
(Southeast Kansas Society)

BACANI, OSWALDO, PO BOX 576,66736
378-3700
44 M 74810 70 GS
BAYLES, HUGH G, PO BOX 30,66736
378-3412
25 M 1902 52 FP
BEAL, RAYMOND J, 600 MADISON,66736
378-2159
12 M 1902 38 GS
RINDT, PHIL, 432 N SEVENTH,66736
378-2298
45 M 1902 71 FP
SUMNER, RALPH N, 712 MADISON,66736
378-2311
31 M 1902 57 FP

FT. LEAVENWORTH
(Leavenworth County Society)

CHAVALA, SUDARSAN, EYE CLINIC,66027
684-4157
43 M 49511 68 OPH

GARDEN CITY—316
(Southwest Kansas Society)

ARROYO, ZEFERINO, 2124 ANTLER RIDGE DR,67846
872-2187
M 74802 GS
AUSTIN, JOHN O, 601 N 6TH,67846
276-2346
14 M 1501 40 FP
BARNARD III, JAMES A, 1601 HARDING,67846
275-9671
31 M 4812 62 OBG
BEGGS, DAVID F, BOX 1077,67846
275-9671
39 M 1902 64 IM
BIGLER, F CALVIN, 801 N FIFTH,67846
275-2141
31 M 801 57 GS
BRUNO, JAMES W, 1133 KANSAS PLAZA,67846
276-8201
42 M 4706 66 FP
CALBECK, JOHN, 2603 BELMONT PLACE,67846
275-9671
50 M 1902 75 IM
COLLINS, SHARON A, 603 N FIFTH,67846
275-9671
51 F 2512 78 PO
CORONADO, EFRAIN, 1001 LABRADOR,67846
275-6111
37 M 25401 55 OR
EICHHORN, FRANK O, BOX 719,67846
276-8132
25 M 1902 56 FP
FENTON, ROBERT M, 603 N FIFTH,67846
275-9671
20 M 1902 54 FP
FRY, LUTHER L, ST CATHERINE HOSP,67846
275-7248
41 M 1902 67 OPH
GARDINER, TED M, 603 N FIFTH,67846
275-9671
M PO
GILBERT II, JOHN H, BOX 1077,67846
275-9671
46 M 1902 70 ORS
GREENWOOD, JAMES F, PO BOX 419,67846
356-1261
33 M 1611 65 FP
HANSON, FRANK W, 603 FIFTH,67846
275-9671
49 M 1902 76 PM
KALBAC, RICHARD W, BOX 1077,67846
275-9671
45 M 2803 70 OBG
KOKSAL, TOM, PLAZA MED CENTER,67846
276-8201
M 1902 FP
MAXFIELD, RUSSELL J, 611 MAGNOLIA DR,67846
276-3342
16 M 1902 41 FP
MEYERS, STEPHEN, BOX 1077,67846
275-9671
48 M 2834 70 PO
MILLER, ROBERT E, BOX 1077,67846
275-9671
26 M 4812 55 GS
RODRIGUEZ, PAUL L, BOX 1729,67846
275-6111
39 M 4706 66 R
SHUSS, JOHN L, 901 N FIFTH,67846
275-2141
49 M 1902 75 GS
SPIKES, MARION E, 603 N 5TH,67846
275-9671
26 M 1902 62 FP
TEARE, MAX E, 1007 DAVIS,67846
276-7689
28 M 1902 54 P
TURNER, JOHN W, 210 E SPRUCE,67846
276-3292
13 M 1902 39 FP
VACHAL, EVA, 609 N FIFTH,67846
275-6111
F PATH
WELCH, MAURA S, BOX 1077,67846
275-9611
50 F OBG
WILEY, HORACE M, BOX 1768,67846
376-6901
12 M 2802 40 GS
ZELLER, MYRON J, BOX 1077,67846
275-9671
38 M 1902 64 OM

GARDEN PLAIN—316
(Sedgwick County Society)

LIND II, EDWARD J, 728 BIERMANN,67050
-
53 M 1902 78 FP
REINHARDT-WULF, TAISSIA L, PO BOX 273,67050
-
19 F 91302 42 DO

GARDNER—913
(Johnson County Society)

REECE, A THOMEN, 121 HICKORY CIRCLE,66030
884-8711
37 M 1902 63 FP
WHITAKER, MARK A, 427 W MAIN,66030
884-8711
53 M 1902 77 PD

GARNETT—913
(Anderson County Society)

DOUGHERTY, THOMAS M, 117 W 6TH,66032
448-5421
28 M 1902 55 FP
HARRIS JR, CLAIB B, 320 S OAK ST,66032
448-5431
17 M 1902 44 FP
HENDERSON, DAVID V, 117 W SIXTH,66032
448-5421
48 M 1902 79 FP
LEITCH, DAVID A, GARNETT MEDICAL CENTER,66032
448-5421
38 M 1902 63 FP
STEVENS, MILDRED J, 202 W 4TH,66032
448-5454
23 F 1902 47 FP
STEVENS, ROBERT L, 202 WEST 4TH,66032
448-5454
23 M 1902 47 FP

GIRARD—316
(Crawford County Society)

FRIGGERI, ROBERT W, 111 N SUMMIT,66743
724-8723
23 M 1902 51 FP
HALL, WESLEY H, PO BOX 158,66743
724-6154
25 M 1902 57 FP

GLASCO—913
(Cloud County Society)

HARWOOD, CLAJOE J, PO BOX 428,67445
568-2245
25 M 1902 55 FP

GOODLAND—913
(Northwest Kansas Society)

AUSTIN, KENNETH D, 520 MAIN,67735
899-3633
33 M 3005 63 FP
FITZGERALD E JAMES, BOX 871, 67735
899-5695
44 M 77 GP
LONG, LLOYD O, 520 MAIN,67735
877-3305
37 M 1720 63 FP
MCCULLOUGH, ROBERT C, 126 E 10TH BOX 180,67735
899-3633
25 M 702 58 GP
OLSON, CLITUS W, 520 MAIN ST,67735
899-3633
16 M 3005 48 GS

GREAT BEND—316
(Barton County Society)

ALDERSON, THOMAS W, 3520 LAKIN,67530
792-5341
50 M 1902 75 FP
ANDERSON, LYLE B, 1636 CHEROKEE,67530
793-8141
28 M 1902 60 DO
BEAHM, ANOL W, 3923 BROADWAY,67530
793-7827
16 M 1902 43 FP
BEAHM, DONALD E, MED ARTS BLDG,67530
792-3625
45 M 1902 71 OPH
BROWN, C REIFF, 1031 JACKSON,67530
792-4391
31 M 3901 57 ORS
CAVANAUGH, CLAIR J, C K M C,67530
792-2617
23 M 1803 47 R
DEGNER, JAMES B, 3515 BROADWAY,67530
792-2517
31 M 1902 57 R
ELLEDGE, E FRED, 2222 FOREST,67530
792-3678
38 M 1902 65 U
EVANS, WILLIAM R, 1912 LINCOLN,67530
793-3574
25 M 1902 53 FP
FIESER, CARL W, 3515 BROADWAY,67530
792-2517
45 M 1902 71 R
FLESKE, LEONARD T, 1315 POLK,67530
792-4383
49 M 1902 75 ORS
GATENO, JOSEPH, 1023 JACKSON SQUARE,67530
793-3501
25 M 64901 50 DBG
HILL, LARY MICHAEL, 1017 A JACKSON,67530
793-8141
51 M 1902 77 FP
HOLT, JOHN M, PO BOX 1328,67530
793-8429
35 M 1902 61 IM
JONES, EDWARD L, 3515 BRDADWAY,67530
792-2511
35 M 1902 61 PATH
KING, WILLIAM T, 3421 FOREST,67530
793-3501
35 M 1902 61 OBG
KIRBY, MERLIN G, 3520 LAKIN,67530
793-3091
31 M 1902 56 GS
KRUEGER, HAVEN C, 1023 JACKSON SQUARE,67530
792-2163
32 M 1902 61 PD
MCALLASTER, WENDALE E, 2111 FOREST,67530
793-3591
24 M 1902 54 GS
NIEDEREE, W CURTIS, 3520 LAKIN,67530
793-3091
30 M 3006 56 GS
POLSON, ROBERT C, BOX A 1422 POLK ST,67530
793-8414
17 M 1902 42 DPH
PRESTON, RICHARD, PO BOX 1328,67530
793-8426
42 M 1902 69 IM
REPLOGLE, CHARLES B, 2111 FOREST,67530
793-3591
27 M 1902 53 FP
RUIZ, CARLOS M, PO BOX 1348,67530
792-3210
25 M 27501 52 P
SAYLER, JEROME, CENTRAL KS MEDICAL CENTER,67530
792-2511
20 M 4113 50 PATH
SCHUETZ, PERRY N, 1422 POLK BOX A,67530
793-8414
45 M 1902 71 OPH
SCHUKMAN, JAY S, 3520 LAKIN,67530
792-5341
50 M 1902 75 FP
SHIVEL, DAVID G, 3523 FOREST,67530
793-3523
28 M 1902 55 FP
SMITH, PERRY MILTON, 3520 LAKIN,67530
792-5341
52 M 1902 77 FP
SWAN, MAJOR MARTIN, 3923 BROADWAY,67530
792-4540
06 M 1902 43 IM
UNREIN, ROBERT J, 1017A JACKSON,67530
792-2504
29 M 1902 58 FP
WHITE, CHARLES _, 2412 DOVE TERRACE,67530
-
06 M 1902 36 DO

WIGGS, JAMES W, 1027 JACKSON,67530
792-1336
36 M 1720 63 N

GREENSBURG—316
(Iroquois County Society)

BRADLEY, J RODERICK, 502 SOUTH WALNUT,67054
723-2127
23 M 1902 47 FP
WALDORF JR, MELVIN H, BRADLEY-WALDORF CLINIC,67054
723-2127
23 M 1902 47 FP

HALSTEAD—316
(Harvey County Society)

AILLON, ALEJANORO J, HERTZLER CLINIC,67056
835-2241
39 M 26402 63 TS
BAILEY, COLIN, HERTZLER CLINIC,67056
835-2241
33 M 35205 59 GYN
BEUGELSDIJK, HENRY PETER, 421 SPRUCE,67056
835-2241
49 M 1902 74 ANES
BOUDREAUX, VELTIN J, PO BOX 53,67056
835-2241
37 M 4812 64 R
BURNETT, A DEAN, HERTZLER CLINIC,67056
835-2241
21 M 1902 52 GS
DECKER, DONALD D, HERTZLER CLINIC,67056
835-2241
31 M 1902 56 CO
EASTES, GARY DEAN, HERTZLER CLINIC,67056
835-2241
44 M 4812 71 U
GNAU, FREDRIC B, 803 MAIN,67056
835-2241
42 M 1902 68 OTO
HAIRE, WILLIAM O, 901 MAIN,67056
835-2241
49 M 1902 74 IM
HARMS, WILMER A, THE HERTZLER CLINIC,67056
835-2241
22 M 1902 56 OPH
HOOFER, WILFORD D, HERTZLER CLINIC,67056
835-2241
30 M 1902 55 TS
MALONE, EUGENE M, HERTZLER CLINIC,67056
835-2241
23 M 1902 56 IM
MARSH, CONNIE M, HERTZLER CLINIC,67056
835-2241
47 F 1902 75 IM
MONTGOMERY, LLOYD DAN, HERTZLER CLINIC,67056
835-2241
43 M 3601 69 P
MONTGOMERYSHORT, RUTH G, HERTZLER CLINIC,67056
—
10 F 1902 37 ENT
MORTON, JOHN E, 320 WALNUT,67056
—
99 M 35211 26 OD
RESCHLY, RON, 603 CHESTNUT, 67056
825-2241
RIZZA, ROBERT G, RT #2,67056
—
30 M 1201 56 PD
SHAH, SHARFUDDIN, HERTZLER CLINIC,67056
835-2241
31 M 70401 58 IM
STOFFER, ROBERT P, HERTZLER CLINIC,67056
835-2241
26 M 1902 48 IM
TEJANO, NEONILO A, HERTZLER CLINIC,67056
835-2241
43 M 74808 67 ORS
WELCH, JACK W, HERTZLER CLINIC,67056
—
18 M 1902 51 OD
WILSON, H RANDOLPH, HERTZLER CLINIC,67056
835-2241
20 M 4112 45 GYN

HANOVER—913
(Northeast Kansas Society)

WARREN, LINDA D, BOX 38,66945
325-2240
44 F 1902 70 FP
WARREN, ROGER O, BOX 38,66945
337-2214
31 M 1902 57 GS

HARPER—316
(Tri-County Society)

BELLAR, RALPH E, 1019 CENTRAL,67058
896-3131
31 M 3005 60 FP
FORREO JR, WALTER A, 121 E MAIN,67058
896-3661
43 M 1902 69 FP
GARONER, BILLIE L, 121 E MAIN,67058
896-3661
25 M 1902 57 FP

HAYS—913
(Central Kansas Society)

ALLEN, JAMES E, 2707 VINE #6,67601
628-3261
46 M 1902 72 IM
APPLEGATE JR, FRANCIS R, 1010 DOWNING,67601
628-8218
30 M 1902 55 OPH
ARTMAN, JOHN C, 2005 LINCOLN DRIVE,67601
625-2518
24 M 2002 48 ANES
BULA, RALPH E, 3209A WILLOW,67601
—
12 M 1902 37 DO
CARLSON, EARL V, DRAWER 430,67601
628-8221
31 M 3005 56 ORS
CECIL III, JOHN, BOX 833,67601
625-6521
43 M 4804 69 R
CODY, DOROTHY, 2704 WOODROW CT,67601
628-4000
29 F 3607 53 ?
CODY, JOHN, 2704 WOODROW CT,67601
628-2871
25 M 401 60 P
COX, ROBERT H, 2507 CANTERBURY RD,67601
628-3051
43 M 1902 70 PD
CRAMM, RUSSELL E, 3004 BROADWAY,67601
625-2518
30 M 1902 56 GS
DYCK, ERIC LEE, FAMILY PRACTICE CLINIC,67601
628-6151
52 M 1902 77 FP
EDDY, VICTOR M, 105 W 13TH,67601
525-2551
29 M 1902 55 GS
ENBERG, ROBERT, 2507 CANTERBURY,67601
628-3051
43 M 1902 69 PD
HAIGLER, JAMES P, 234 WEST 11TH,67601
625-2537
13 M 3006 39 FP
HALLING, L WILLIAM, 1300 EAST 13TH,67601
625-5646
27 M 5002 57 PATH
HANSA, HANSA, 2503 CANTERBURY,67601
628-3245
45 M 89101 69 OBG
HUTCHISON, GLEN C, 3200 COUNTRY LANE,67601
628-8251
21 M 1902 50 ANES
KANE JR, WILLIAM M, CANTERBURYS CLINIC PA,67601
628-3245
27 M 1001 54 OBG
KIFER, C JAMES, BOX 833,67601
625-6521
45 M 1902 71 OR
LASLEY, MICHAEL B, 2707 VINE SUITE 7,67601
628-3217
45 M 1902 71 GS
MATTICK, IRVIN H, 2818 N VINE,67601
628-8221
18 M 2802 43 ORS

MEDINA, ANIBAL, 2707 VINE,67601
628-1019
34 M 45101 62 U
MYRICK, MICKEY, 2501 CANTERBURY RD,67601
628-6151
42 M 3005 74 FP
NEIL, ROY NEWTON, 1108 MAIN ST S 201B,67601
628-3215
38 M 300S 65 PATH
NEWCOMB, WARD M, 1300 E 13TH,67601
625-5646
47 M 300S 71 PATH
NIELSEN, MARY L, 1300 E THIRTENTH,67601
625-5646
47 F 1902 77 PATH
RAJEWSKI, RICHARD L, 2509 CANTERBURY,67601
628-6151
51 M 1902 76 FP
REYNOLDS, JEFFREY C, 2517 CANTERBURY RD,67601
625-7311
39 M 1902 64 ENT
REYNOLDS, LLOYD W, 2110 ASH,67601
-
10 M 3840 34 OD
RICHARDS, DALLAS LEE, 2209 CANTERBURY RD STE A,67601
625-4224
49 M 1902 76 IM
ROBINSON, MICHAEL D, 2209 CANTERBURY RD,67601
625-4224
50 M 3005 76 IM
RUTNGAMLUG, LUECHA, 3004 BROADWAY,67601
628-6175
40 M 89101 68 GS
SILER, EUGENE T, 1010 DOWNING,67601
628-8218
24 M 1902 52 OPH
STADALMAN, ROSS EUGENE, 2707 VINE SUITE 7,67601
628-3217
47 M 1902 73 GS
STUMP, HARL G, 2717 CANAL BLVD,67601
628-2825
39 M 1902 65 GS
WATTS, HARRY E, 1010 DOWNING AVE,67601
628-8218
27 M 702 54 OPH
WEBER, WALLACE N, 2707 VINE SUITE 10,67601
628-3231
43 M 1902 69 D
WERTH, DARRELL DEAN, 2209 CANTERBURY RD STE D,67601
628-6014
50 M 1902 75 U
WIEGMAN, HUGH ALAN, BOX 833,67601
625-6521
34 M 1803 60 R
WILCOX JR, HOWARD L, PO DRAWER 430,67601
628-8221
44 M 1902 70 ORS

HAYSVILLE—316
(Sedgwick County Society)

MORGAN, NOVA L, 301 W GRAND,67060
225-1371
20 M 3901 50 FP

HERINGTON—913
(Dickinson County Society)

BUSTOS, JONAS G, 1005 NORTH B,67449
258-2215
41 M 74810 68 GS
DOZIER, FREO S, 1005 NORTH B STREET,67449
258-2215
10 M 4804 34 FP

HESSTON—316
(Harvey County Society)

DIENER, CLAYTON H, PO BOX 386,67062
327-4122
18 M 1902 54 GS
FRIESEN, FLORENCE V, SHOWALTER VILLA,67062
-
87 F 1611 14 OD
YODER, VERNON E, ROUTE #1,67062
283-2400
31 M 4802 61 P

HIAWATHA—913
(Northeast Kansas Society)

BASSETT, P MARCUS, 314 OREGON,66434
742-2161
51 M 1902 77 FP
DUCKETT, THOMAS G, 201 MIAMI,66434
-
10 M 1902 34 OD
HALL JR, ERNEST B, 314 OREGON,66434
442-3341
53 M 1902 78 FP
LARSON, DELBERT L, 314 OREGON,66434
742-2161
30 M 1803 64 FP
MEIDINGER, RAY, 111 S FOURTH,66434
742-2135
03 M 3005 32 FP
SINNING, GARY, 314 OREGON,66434
742-2161
49 M 1902 74 FP

HIGHLAND—913
(Northeast Kansas Society)

CORDER, ROBERT L, ,66035
442-3341
23 M 1902 49 FP

HILL CITY—913
(Central Kansas Society)

REDDY, B N, 114 E WALNUT,67642
674-2255
38 M 49557 66 PD
REDDY, P JAGANNADHA, 80 WALNUT DRIVE,67642
674-2255
42 M 49511 66 GS

HILLSBORO—316
(Marion County Society)

ENS, GERHARD GEORGE, 613 SOUTH MAIN,67063
947-5931
20 M 1902 55 FP
ENS, PETER, 209 SOUTH MAIN,67063
947-3671
14 M 1902 51 FP
FRANZ, ROBERT G, 704 S MAIN,67063
947-3197
33 M 3901 59 FP
LOEWEN, PETER S, SKILLED NURSING UNIT,67063
-
91 M 1902 30 OD

HOISINGTON—316
(Barton County Society)

FERNANDEZ, HECTOR O, 351 W TENTH,67544
653-4944
41 M 74809 66 GS
MOORE, ROBERT, 814 NORTH ELM,67544
653-2151
22 M 3901 53 FP

HOLTON—913
(Jackson County Society)

MOSER, ERNEST C, 438 HILLCREST DR,66436
-
09 M 1902 34 OD
MOSER, M ROSS, 418 WEST FIFTH,66436
364-3116
19 M 1902 47 FP
MOSER, ROY H, 801 IOWA,66436
-
04 M 1902 32 OD
SEELEY, JAMES C, 418 WEST 5TH,66436
364-3116
34 M 1902 64 FP

HORTON—913
(Northeast Kansas Society)

FRANCISCO, EDGARDO, PO BOX 6,66439
486-2646
39 M 74802 57 GP
WALTON, PHILIP D, 300 W 18TH,66439
486-2828
32 M 1902 63 FP

HOXIE—913
(Northwest Kansas Society)

NEUENSCHWANDER, JOHN, 700 MAIN,67740
675-3292
26 M 2802 51 FP
NEUENSCHWANDER, JOHN RAND, 700 MAIN,67740
675-3292
47 M 1902 72 FP

HUGOTON—316
(Seward County Society)

FREDERICK, M F, 1006 S JACKSON,67951
544-2784
20 M 1902 44 FP
IHRIG, ROGER W, 1006 S JACKSON,67951
544-2784
49 M 1902 74 FP
LENEVE, ROBERT T, 1006 S JACKSON,67951
544-2784
21 M 3901 46 FP

HUMBOLDT—316
(Southeast Kansas Society)

LONG, EDWARD E, 8TH & NEW YORK,66748
473-2411
21 M 1902 50 FP

HUTCHINSON—316
(Reno County Society)

ADAMS JR, MARCUS W, 2101 N WALDRON,67501
663-6121
33 M 3901 59 PD
ALBRIGHT, JEROLD D, 2101 N WALDRON,67501
663-6121
39 M 1902 66 FP
BAUER, THOMAS A, 2101 N WALDRON,67501
663-6121
41 M 1902 67 IM
BLANK, JOHN N, ROUTE 5 BOX 220,67501
-
07 M 1902 38 DD
BORRA, MARID J, 1 WEST 8TH,67501
662-1751
24 M 2401 47 U
BOS, NORMAN C, 2101 N WALDRON,67501
663-4406
24 M 1611 47 ORS
BOUDA, DAVID W, 2101 WALDRON,67501
663-6121
45 M 1902 72 IM
BRADA, DONALD ROBERT, 308 W 20TH,67501
662-5041
39 M 1902 65 P
BURGER, J DALE, 2101 N WALDRON,67501
663-6121
21 M 1902 46 FP
CASADY, GILBERT N, 39 LINKSLAND DR,67501
663-3657
23 M 1902 53 ANES
CASEY, JAMES, 2101 N WALDRON,67501
663-6121
42 M 3005 69 PD
CHERVEN, PHILIP L, 1100 N MAIN,67501
662-3364
45 M 2501 71 PD
DEPENBUSCH, FRANCIS L, 1708 E 23RD,67501
663-7187
38 M 1902 65 OPH
ECKART, DE MERLE E, 312 WOLCOTT BLDG,67501
662-4921
14 M 1902 40 P

FALTER, RICHARD T, 1708 E 23RD ST,67501
663-7187
38 M 1902 67 OPH
FOSS, DANIEL C, 2101 N WALDRON,67501
663-6121
43 M 1902 69 GE
GOODPASTURE, WILLARD C, RR 3 BOX 45,67501
-
10 M 1602 36 DD
GRAVES, KATHRYN, 2101 N WALDRON,67501
663-6121
49 F 1902 74 D
HEDRICK, KENNETH E, 2101 N WALDRON,67501
663-6121
27 M 1902 53 GS
HOLDERMAN, WALLACE D, 2101 N WALDRON,67501
663-4406
28 M 1902 54 ORS
JARROTT, JOHN B, 1100 N MAIN,67501
663-2151
16 M 1902 40 ORS
JOHNSON, RANDLE C, 1100 N MAIN,67501
663-2151
46 M 1902 72 IM
KEPKA, DENNIS, 3117 S MEADOWLAKE DR,67501
663-2151
43 M 56101 77 FP
KLOSTERHOFF, BRUCE E, 1701 E 23RD,67501
663-2108
45 M 1902 71 P
LESSER, DANE A, 2101 N WALDRON,67501
663-6121
49 M 3901 75 U
LETTNER, HANS T, BOX 159,67501
662-7801
23 M 40716 49 PATH
LUKENS, DAVID, 2101 N WALDRON,67501
663-1136
24 M 2307 48 IM
MCCOY, CHARLES T, 310 WOLCOTT BLDG,67501
662-0121
16 M 1902 41 OPH
MCMULLEN, JOSEPH E, 2101 N WALDRON,67501
663-6121
33 M 1902 62 GS
MULL, JOHN C, 2101 N WALDRON,67501
663-6121
34 M 1902 61 OBG
NEEL, WILBUR B, 2101 N WALDRON,67501
663-6121
24 M 1902 59 IM
NEUSCHAFER, DARREL R, 2101 N WALDRON,67501
663-6121
48 M 1902 74 OBG
NUNEMAKER, MARION E, RR 5 BOX 15,67501
662-5391
21 M 1902 46 ANES
OPENSHAW, CALVIN R, 1824 N MAIN,67501
662-3305
21 M 4901 44 GS
PAINE, GEORGE E, 220 W 23RD,67501
-
94 M 1606 19 DO
PEASE, GARY L, 521 WILEY BLDG,67501
662-4458
41 M 3005 67 OTO
PERKINS, JACK L, 2101 N WALDRON,67501
663-6121
24 M 1902 53 FP
RATE, PEGGY S, 2221 E 56TH,67501
663-6121
46 F 1902 73 PD
RATE, ROBERT R, 2221 E 56TH,67501
663-6121
47 M 1902 74 IM
SAVAGE, WILLIAM R, 1100 N MAIN,67501
663-2151
48 M 3901 74 IM
SCALES, WILLIAM M, PLAZA TOWERS,65611
-
00 M 1803 31 DD
SCHEEL, BRADLEY J, 1100 N MAIN,67501
663-2151
48 M 1902 74 IM
SCHROLL, JACK C, 2101 N WALDRON,67501
663-6121
24 M 1902 49 OBG
SHAW JR, JAMES W, BOX 1646,67501
662-7801
40 M 1902 65 PATH
SHEARS, ROBERT N, 1100 N MAIN,67501
662-3364
20 M 1902 44 PD

SMITH, THOMAS WILLIAM, 521 WILEY BLDG,67501
662-4458
43 M 1643 68 OTO
SPITZER, JEROME S, 1100 N MAIN,67501
663-2151
33 M 3005 59 FP
STAFFORD, ROBERT W, 2101 N WALDRON,67501
663-6121
43 M 2101 69 IM
STEINMETZ, WM, 1701 E 23RD,67501
663-6811
51 M 2802 77 PATH
STENSAAS, CARL O, 401 WOLCOTT BLDG,67501
663-9147
10 M 1902 38 D
STOUT, JAMES M, 2101 N WALDRON,67501
663-6121
29 M 1902 55 FP
SUMNER, JOYCE R, 3011 - B NUTMEG LANE,67501
662-0822
26 F 1902 51 ANES
SUMNER, MARION M, 3011-B NUTMEG LN,67501
662-0822
25 M 1902 52 IM
TAYLOR, E J, 1100 N MAIN,67501
663-2151
34 1902 61 FP
TISDALE, TERRANCE C, HUTCHINSON CLINIC PA,67501
663-6121
36 M 5701 61 ORS
TWEITO, DAVID H, 1100 N MAIN,67501
662-3364
38 M 1803 64 PD
VON RUDEN, WILLIAM J, 2100 N WALDRON,67501
662-3306
26 M 1611 52 GS
WEIDENSAUL, D N, 2101 N WALDRON,67501
663-6121
50 M 1902 75 IM
WILLIAMSON, JOHN, 1100 N MAIN,67501
669-0191
35 M 2501 60 ORS
WORTMAN, JACK A, 2101 N WALDRON,67501
663-6121
34 M 1902 62 IM

INDEPENDENCE—316
(Southeast Kansas Society)

BAIR, ALBERT E, PO BOX 925,67301
331-7650
15 M 1902 44 GS
BARBERA, PORTER E, 800 WEST CHESTNUT,67301
331-4400
19 M 4707 46 FP
BEAHM, EDGAR H, 307 PROFESSIONAL BLDG,67301
331-2311
13 M 1902 44 FP
CHAPPUIE, WILLIAM G, 800 CHESTNUT WEST,67301
331-5440
24 M 1902 51 FP
EMMOT, WILLIAM W, 800 WEST CHESTNUT,67301
331-6350
45 M 1902 71 IM
EMPSON, CHARLES L, 400 N 14TH,67301
331-6019
37 M 1902 68 FP
GAREY, WILLIAM JOHN, PO BOX 388,67301
331-2200
44 M 6501 70 DR
KNUTH, KENNETH L, 2900 TERRA VISTA,67301
331-2200
22 M 1902 50 R
MYERS JR, EARL B, BOX 548,67301
331-3420
32 M 2803 64 GS
OSBORN DO, ROBERT M, PO BOX 872,67301
331-2070
M 2843 69 GP
ROBINSON, EDGAR L, 209 NORTH 6TH,67301
331-1450
15 M 1902 42 FP
SHEIKH, MASOOD A, 900 W MYRTLE,67301
331-2803
44 M 70402 66 GS
SUTTON, ROBERT E, PO BOX 1003,67301
331-7700
46 M 1902 72 FP

IOLA—316
(Allen County Society)

COPENING, TELL B, 305 N WASHINGTON,66749
365-2134
43 M 1902 69 FP
DETAR, GEORGE F, 219 W MADISON,66749
365-5175
28 M 1902 57 GS
DICK, WILLIS G, BOX 826,66749
365-2131
13 M 512 41 GS
HANSON, DAVID C, 501 E MADISON BOX 714,66749
365-2131
46 M 512 73 FP
LENSKI JR, FRANCIS X, 206 S JEFFERSON,66749
365-3901
26 M 1506 49 FP
MYERS, W EUGENE, 211 S STREET,66749
365-3732
12 M 1902 46 FP
PEES, GERALD B, 219 W MADISON,66749
365-5175
15 M 1902 43 GS
SCHMAUS, LYLE F, 202 E STREET,66749
-
99 M 1803 26 OO

JETMORE—316
(Ford County Society)

O'SHEA, JAMES G, BOX 545,67854
357-8321
18 M 3901 48 FP

JEWELL—913
(Mitchell County Society)

PLOWMAN, CARL W, ,66949
-
99 M 1506 26 FP

JOHNSON—316
(Southeast Kansas Society)

DAILEY, RONALD F, BOX 470,67855
492-6230
31 M 1902 57 FP

JUNCTION CITY—913
(Geary County Society)

BOLLMAN, CHARLES S, PO BOX 397,66441
762-4575
41 M 3901 66 GS
BRETHOUR, LESLIE J, 1106 ST MARYS RD,66441
238-4151
13 M 3006 39 FP
BUNKER JR, HERBERT L, 1106 ST MARYS RD,66441
238-5131
20 M 1606 45 FP
COPELAND, GARY A, GEARY COMMUNITY HOSP,66441
762-2387
42 M 1902 68 R
CRAIG, THOMAS A, 1106 ST MARYS RD STE 102,66441
762-4255
53 M 1902 78 IM
FIELDSTONE, PAUL R, 1106 ST MARYS RD STE 204,66441
762-4300
46 M 6501 71 U
LABHSETWAR, S A, MEDICAL ARTS BLDG,66441
762-4147
39 F 49528 64 OBG
MACE, RONALD D, PO BOX 163,66441
762-4884
42 M 3901 74 FP
MINNICK, CHARLES V, 602 NORTH JEFFERSON,66441
-
08 M 2105 35 FP
O'DONNELL, HARRY E, 1106 ST MARYS RD,66441
762-4947
14 M 4113 42 FP
PATEL, MAHENDRA N, 1106 ST MARYS RD STE 206,66441
762-2327
48 M 35207 74 IM
SCOTT, ALEX, 507 WEST 6TH,66441
238-2518
23 M 5605 48 FP
WRIGHT, STANLEY E, 1106 ST MARYS RD,66441
762-4884
47 M 3901 74 FP

KANSAS CITY, KANSAS—913
(Wyandotte County Society)

A8DOU, NABIH I, KU MEDICAL CENTER,66103
588-6586
34 M 33002 58 A
ALEXANDER, CHARLES E, 600 NEBRASKA,66101
321-6670
43 M 401 70 OBG
ALEXANDER, CLYDE W, 1514 NORTH 5TH,66101
281-4380
96 M 4707 23 FP
ALGIE, WILLIAM H, 907 N 7TH,66101
371-5079
02 M 1902 27 IM
ALLEGRE, ANN, 1225 N 78TH,66112
788-7099
50 F 1902 77 IM
ALLEN, TIMOTHY E, 9201 PARALLEL PKWY,66112
334-4110
49 M 1902 R
ALLEN, WILLIAM R, 9201 PARALLEL PARKWAY,66112
334-4110
22 M 1902 46 R
AMARE, MAMMO, KU MEDICAL CENTER,66103
588-6031
36 M 60501 61 IM
ARAKAWA, KASUMI, KU MED CENTER,66103
588-6670
26 M 57211 53 ANES
ARENAL, ANGELA C, BETHANY HOSPITAL,66102
281-8400
38 F 35204 60 ANES
A5HER, MARC A, K U MED CENTER,66103
588-6130
36 M 1902 62 ORS
ATKINS JR, FLOYD L, K U MED CENTER,66103
588-6015
43 M 5104 69 CD
8AILIE, MICHAEL DAVID, KU MED CENTER,66103
355-6339
36 M 1720 64 PNP
8ASINGER, BRADLEY B, 3824 BOOTH APT 3,66103
53 M 1902
BAS5 JR, LEWIS N, 1975 N 5TH,66101
321-2320
21 M 1902 45 PD
8ATNITZKY, SOLOMON, K U MED CENTER,66103
588-6835
40 M 83601 64 DR
8ECKER, LESLIE E, 600 NEBRASKA,66101
342-4010
23 M 1003 46 U
8ERGIN, JAMES J, 8ETHANY MED CENTER,66102
281-8767
28 M 2407 54 IM
8ICHLMEIER, FRANKLIN G, 155 S 18TH,66102
371-6800
33 M 1902 58 CDS
BIGONGIARI, LAWRENCE R, KU MED CENTER, 66103
588-6800
44 M 1611 69 DR
8ILLINGSLEY, THAD H, 155 S 18TH,66102
321-3844
41 M 1902 66 P
8IXLER II, THOMAS J, KU MED CTR CARDIOVAS SURG,66103
588-6107
47 M 1205 74 CDS
8ODENSTEINER, DAVID C, K U MED CENTER,66103
588-6077
51 M 1803 76 IM
8OGGAN, MICHAEL D, 155 S 18TH,66102
371-6800
40 M 1201 67 CDS
8OLINGER, ROBERT E, KU MEDICAL CENTER,66103
588-6022
19 M 1902 43 END
BOLTON, VICTOR E, 155 SOUTH 18TH,66102
371-2020
24 M 1902 48 OBG
8OSILEVAC, FREO N, 155 S 18TH,66102
16 M 1902 44 OPH
8RACKETT JR, CHARLES E, KU MED CENTER,66103
588-6117
20 M 3501 44 NS
8RILLHART, MAXINE T, 1610 WASHINGTON BOULEVARD,66102
321-4800
15 F 1902 50 FP
8ROOKS, WILLIAM HENRY, 155 S 18TH,66102
371-4343
49 M 1902 74 R
8ROWN, JOHN V, 3728 CAM8RIDGE,66103
432-5610
58 M 1902
8UBB, STEPHEN K, 155 S 18TH,66102
371-6802
48 M 1902 74 ORS
8URGER, WILLIAM E, 355 NEW BROTHERHOOD BLDG,66101
371-1017
21 M 3006 51 GS
BUTTERFIELD, MARI ANNE, KU MED CENTER,66103
588-6670
52 F 1902 77 ANES
CALDERON, JAIME, 4631 ORVILLE SUITE 201,66102
596-1185
39 M 25401 66 IM
CAMERON, WILLIAM J, KU MEDICAL CENTER,66103
588-6200
29 M 2501 54 OBG
CARLIN, JAMES WILLARD, KU MED CENTER,66103
588-6670
51 M 1902 76 ANES
CARPENTER, PAUL R, 155 SOUTH 18TH,66102
371-6800
24 M 1902 50 TS
CHALIAN, ALEXANDER R, 2648 MINNESOTA AVENUE,66102
03 M 3509 37 OO
CHANG, C H JOSEPH, KU MED CENTER,66103
588-6807
29 M 58301 53 R
CHIN, TOM D, KUMC - HUMAN ECOLOGY DEPT,66103
588-7175
22 M 2501 43 ID
CHO, CHENG T, K U MED CENTER,66103
588-6336
37 M 38501 62 PD
CHONKO, ARNOLD M, KU MED CENTER,66103
588-6969
43 M 3840 69 NEP
CHRYSANT, STEVEN G, KU MED CENTER,66103
861-4700
34 M 41801 59 CD
CHUN, CHUNG S, K U MED CENTER,66103
588-6324
30 F 58301 53 PD
COALE, LLOYD H, 5020 GREELEY,66104
13 M 1902 43 OO
COOK, JAMES O, KU MED CENTER,66103
588-6031
36 M 6505 60 HEM
COOKE, ALLAN R, KUMC DEPT OF MED,66103
588-6990
36 M 14303 58 GE
COONFIELD, JAMES W, 1428 S 32ND,66106
384-1630
45 M 1902 78 FP
COX III, IRA L, 155 SOUTH 18TH,66102
371-4343
43 M 1902 68 DR
COX JR, WALLACE F, 4631 ORVILLE,66102
287-3600
30 M 1902 56 GS
CROCKETT, CHARLES A, 155 S 18TH,66102
342-2200
19 M 401 44 OPH
CULP, LOUIS M, 1645 WASHINGTON BLVD,66102
371-1077
24 M 1902 53 FP
CURTIS, MIRJANA R, KU MED CENTER - OB/GYN,66103
588-6237
46 F 95702 69 OBG
DARR, RICHARD B, 320 TERRACE TR WEST,66106
676-2097
42 M 3401 70 IM
DAVIS, CHRISTOPHER G, 219 HURON BLDG,66101
321-9313
09 M 1902 39 FP
DAY, HUGHES W, BETHANY MED CENTER,66102
281-8856
15 M 1902 39 IM
DE SMET, ARTHUR AUGUST, KU MED CENTER - DIAG RAD,66103
588-6800
47 M 2501 72 DR
DEITZ, MICHAEL R, 155 S 18TH,66102
342-2222
32 M 4101 58 OPH
DEMOTT, WAYNE R, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
34 M 4002 59 PATH
DIALLO, GASTON I, 600 NEBRASKA,66117
281-2888
35 M 86905 64 GE

DICK, ARTHUR R, KU MEDICAL CENTER,66103
588-6985
34 M 2301 65 N
DIEDERICH, DENNIS A, K U MEDICAL CENTER,66103
588-6981
36 M 2834 61 1M
DONNELLY, F MICHAEL, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
48 M 1902 74 ANES
DRAINE, QUIDA D, KU MED CENTER AMBUL CARE,66103
588-3974
48 F 2802 75 1M
DUJOVNE, CARLOS A, K U MED CENTER,66103
588-6026
37 M 13201 61 CP
DUNN, MARVIN I, KU MED CENTER,66103
588-6015
27 M 1902 54 CD
EGEA, FERNANDO M, 8919 PARALLEL PARKWAY,66112
334-3400
37 M 13206 62 N
ENRIQUEZ JR, ROMAN S, 510 SOUTHWEST BLVD,66103
262-6068
48 M 74801 72 1M
FABIAN, CAROL J, K U MED CENTER,66103
588-6029
46 F 1902 DN
FITZPATRICK, M ROBERT, 1610 WASHINGTON BLVD,66102
321-4800
20 M 1803 44 FP
FLOERSCH, HUBERT M, 155 S 18TH,66102
371-2020
08 M 1902 35 OBG
FORET, JOHN D, KU MED CENTER,66103
588-6147
26 M 1602 53 U
FOX, DEANNA K, K U MED CENTER,66103
588-6670
48 F 1902 74 ANES
FOX, HOWARD A, K U MED CENTER,66103
588-6337
33 M 3501 62 PD
FRANCISCO, W DAVID, 155 S 18TH,66102
371-6802
21 M 1902 44 ORS
FRIESEN, STANLEY R, KU MED CENTER,66103
588-6108
18 M 1902 43 GS
GERJARUSAK, PRAPAS, 8919 PARALLEL SUITE 208,66112
788-9604
46 M 89101 71 1M
GILHOUSEN, FREDERIC M, 1029 N 32ND,66102
281-5252
40 M 1902 66 ORS
GIRON JR, LOUIS T, K U MED CENTER DEPT OF N,66103
588-6970
43 M 2802 68 N
GLOERSEN, PETER F, 3807 BOOTH,66103
588-
57 M 1902
GODFREY, WILLIAM A, KU MED CENTER,66103
588-6600
38 M 1902 65 OPH
GOERTZ, KENNETH K, 222 HC MILLER KUMC,66103
588-6311
50 M 1902 75 PD
GOERTZ, LEO R, 155 S 18TH ST,66102
371-4343
22 M 1902 52 R
GOODWIN, DONALD W, KU MED CENTER,66103
588-6402
31 M 1902 64 P
GOTO, HIROSHI, KUMC - ANES DEPT,66103
588-6670
42 M 57241 67 ANES
GRADY, KENNETH L, 155 S 18TH,66102
321-3844
36 M 1902 69 P
GRANTHAM, JARED J, K U MED CENTER,66103
588-6983
36 M 1902 62 NEP
GREENBERGER, N J, KU MEDICAL CENTER,66103
588-6001
33 M 3806 59 1M
GRUENDEL, RICHARD A, 1029 N 32ND,66102
281-5252
29 M 1902 55 ORS
GRUENDEL, VIRGINIA T, 6926 GARFIELD,66102
299-2787
30 F 1902 55 FP
GUARDIA, DAVID K, UKSM-KC, 66103
588-6243
52 M 1902 77 OBG
HALL III, THOMAS BRYAN, KU MED CENTER,66103
588-6400
43 M 2802 69 P
HANCOCK, ALAN C, 9201 PARALLEL,66112
299-1474
35 M 1902 65 FP
HANSON, FREDERICK, K U MED CENTER,66103
588-6044
48 M 506 74 1M
HARA, GLENN S, KU MED CENTER,66103
588-6200
43 M 502 69 OBG
HARDIN, CREIGHTON A, KU MED CENTER,66103
588-6106
18 M 5605 43 GS
HARRIS, JO ANN SPEIGEL, K U MED CENTER,66103
588-2773
50 F 3901 76 PD
HART, KELLY Z, 155 S 18TH,66102
371-4343
50 M 1902 75 OR
HART JR, PAUL V, PO BOX 4187,66104
299-2059
50 M 3006 76 1M
HARTMAN, CHARLES R, K U MED CENTER,66103
588-6111
37 M 1902 66 1M
HARTMAN, GERALD V, KUMC,66103
588-6815
20 M 1902 45 TR
HENNING JR, HAROLD JOHN, 4116 BOOTH,66103
-
55 M 1902
HERMRECK, ARLO S, K U MED CENTER,66103
588-7232
38 M 1902 65 GS
HETTINGER, MICHAEL E, KU MED CENTER,66103
588-6600
46 M 4706 75 OPH
HIEBERT, JOHN M, UKSM-KC, 66103
588-6143
42 M 2405 67 PS
HINTHORN, DANIEL R, KU MED CENTER,66103
588-6035
41 M 1902 67 1M
HITCHCOCK, C THOMAS, 155 SO 18TH SUITE 200,66102
371-2900
M 1902 78 GS
HOADLEY, WILLIAM D, KU MED CENTER-MEDICINE,66103
588-3974
31 M 1902 56 1M
HODGES, GLENN R, K U MED CENTER,66103
588-6035
41 M 1602 67 1D
HOLDCRAFT, JACQUELYNE, 4631 DRVILLE SUITE 203,66102
321-1161
36 F 2105 63 ENT
HOLMES, FREDERICK F, KUMC,66103
588-6987
32 M 5404 57 1M
HOLMES, GRACE E, KUMC,66103
588-6325
32 F 5404 57 PD
HOLMES, JOHN A, 155 S 18TH,66102
-
47 M 1902 77 1M
HOOVER, CYNTHIA J, K U MED CENTER,66103
588-6390
53 F 4112 78 PD
HORTON, WILLIAM A, K U MEDICAL CENTER,66103
588-6043
45 M 1902 71 1M
HUERTER, QUENTIN C, WYANDOTTE MED BLDG STE226,66112
299-8800
31 M 1902 59 OPH
HURWITZ, ARYEH, KU MED CENTER,66103
588-6026
36 M 2802 61 1M
IBARRA, RICHARD C, 754 PACIFIC,66101
342-3969
26 M 64902 57 FP
INGRAM, JOHN E, 1428 S 32ND,66106
384-1630
24 M 3006 56 FP
INNES, ROBERT C, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
25 M 2802 49 R
JACOBS, DAVID S, 8929 PARALLEL PARKWAY,66112
334-2500
31 M 2501 56 PATH
JACOBS, RAE R, K U MED CENTER,66103
588-6163
35 M 3506 62 ORS

JAHANIAN, DARYOUSH, 155 S 18TH,66102
371-2020
40 M 51701 64 OBG
JAYARAM, MARANDAPALLI R, 8919 PARALLEL,66112
492-6200
42 M 49509 65 PD
JEWELL, WILLIAM R, KU MED CENTER,66103
S88-6112
35 M 1611 61 GS
JOHNSON, BRUCE E, KU MED CENTER,66103
S88-6000
50 M 514 76 IM
JOHNSON, CYNDA A, KU MED CTR FAMILY PRACTIC,66103
S88-1908
51 F 506 77 FM
JOHNSON, JOHN E, BETHANY MED CTR,66102
281-8815
17 M 4706 43 PATH
JONES JR, HERMAN H, 600 NEBRASKA,66101
342-4010
25 M 4707 54 GS
KALIVAS, JAMES T, KU MED CENTER,66103
S88-6028
38 M 502 63 D
KARNAZE, MICHAEL G, 1900 WASHINGTON AVE,66102
-
56 M 1902
KENNEDY, JAMES A, K U MED CENTER,66103
S88-6000
35 M 2834 61 IM
KEPES, JOHN J, K U MED CENTER - PATH DEP,66103
588-7075
28 M 47301 52 PATH
KERBY, GERALD R, KU MED CENTER,66103
S88-6044
32 M 1902 58 PUD
KESTENBAUM, THELDA M, K U MED CENTER,66103
S88-6028
48 F 3101 73 O
KETCHERSIDE JR, WILLIAM J, 2918 S 9TH PLACE,66103
-
55 M 1602 79 GS
KIM, JONG M, KUMC,66103
588-6670
40 M 58302 64 ANES
KING, CHARLES R, K U MEDICAL CENTER,66103
588-6200
47 M 1902 71 OBG
KIRCHNER, FERNANDO R, 155 S 18TH SUITE 270,66102
371-7333
30 M 64901 55 OTO
KRANTZ, KERMIT E, KU MED CENTER,66103
S88-6201
23 M 1606 48 OBG
KRUMME, BARBARA J, 3815 SPRINGFIELD #2A,66103
-
59 F 1902
KWEE, SIOE T, PROVIDENCE-ST MGT HLTH CT,66112
334-2500
36 F 1720 63 PATH
KYNER, JOSEPH L, KU MED CENTER,66103
S88-5554
34 M 1902 60 END
LAI, CHI-WAN, K J MED CENTER DEPT OF N,66103
588-6014
44 M 38502 69 N
LAING, ROBERT R, 155 S 18TH ST,66102
371-4301
37 M 1643 61 GE
LANSKY, LESTER L, K U MED CENTER,66103
S88-6300
33 M 2604 65 CHN
LANSKY, SHIRLEY B, K U MED CENTER,66103
588-6400
35 F 2604 60 CHP
LAVIN, MARK J, KU MED CENTER - ANES DEPT,66103
588-6670
51 M 1902 76 ANES
LAWWILL, THEODORE, K U MED CENTER,66103
588-6505
37 M 4705 61 OPH
LAYBOURNE JR, PAUL C, KU MED CENTER,66103
588-6475
19 M 3509 44 CHP
LEE, JAE M, 4631 ORVILLE - SUITE 109,66102
677-3555
40 M 58302 65 GS
LEE, KYO R, KUMC,66103
588-6800
33 M 58302 59 R
LEE JR, JAMES G, 5739 METCALF CT,66202
371-2330
18 M 1902 44 OBG
LEMOINE JR, ALBERT N, K U MED CENTER,66103
588-6600
18 M 2802 43 OPH
LEO, WILLIAM A, K U MED CENTER,66103
588-6109
22 M 1902 48 GS
LEVINE, ERROL, K U MED CENTER,66103
S88-6800
41 M 83601 64 DR
LIEBERMAN, BRUCE IRWIN, KU MED CENTER,66103
588-6300
49 M 3819 74 PD
LIN, JAMES T Y, K U MED CENTER DEPT OF N,66103
588-6042
47 M 38502 72 N
LINDSLEY, CAROL B, K U MED CENTER,66103
S88-5907
41 F 5404 68 PD
LINDSLEY, HERBERT B, K U MED CENTER,66103
588-6008
40 M 1902 66 RHU
LINN, CATHERINE P, 8919 PARALLEL PKWY,66112
788-9797
52 F 1902 77 OBG
LINSHAW, MICHAEL A, K U MED CENTER,66103
588-6300
40 M 4109 66 PD
LIU, CHIEN, K U MED CENTER,66103
S88-6035
21 M 24217 47 IO
LLOYD, HARVEY L, PO BOX 6037,66106
-
08 M 1803 36 FP
LOTITO, CARLOS A, 4631 ORVILLE,66102
596-1185
29 M 13201 56 IM
LOWMAN, JAMES T, KUMC,66103
S88-5701
31 M 401 58 PD
LUKERT, BARBARA P, KU MED CENTER,66103
588-6048
34 F 1902 60 END
LYNCH, SEAN R, KU MED CENTER,66103
S88-6031
38 M 83601 61 HEM
MACARTHUR, RICHARD IAN, KUMC 39TH AT RAINBOW,66103
S88-6100
46 M 1902 73 GS
MAGRINA, JAVIER F, K U MED CENTER OBG DEPT,66103
S88-6244
49 M 84701 68 OBG
MANGOLD, JOEL VOYCE, KU MED CENTER,66103
S88-6670
50 M 1902 76 ANES
MANI, MANI M, KUMC,66103
S88-6136
37 M 49527 60 PS
MANSFIELD, CARL M, K U MED CENTER - RM 1179,66103
S88-7350
28 M 1003 56 TR
MARPLES, BRADLEY W, 3904 ADAMS,66103
-
56 M 1902
MARSH, ALICE GARRISON, K U MED CENTER,66103
S88-6390
24 F 3545 49 PD
MARTIN, JOSEPH P, 9201 PARALLEL,66112
334-1515
49 M 1902 74 IM
MARTIN, NORMAN L, K U MED CENTER,66103
S88-6800
36 M 1902 62 OR
MARTINEZ, JOHN D, KU MED CTR DEPT OF MED,66103
588-6008
48 M 1902 74 IM
MASTERS, FRANCIS W, KU MED CENTER,66103
S88-6142
20 M 3545 45 PS
MATHEWSON, HUGH S, KUMC,66103
S88-6675
21 M 1902 44 ANES
MATTIDLI, LEONE, KUMC,66103
S88-6311
32 M 56115 56 PDC
MC PHEE, MARK S, KU MED CENTER-MEDICINE,66103
S88-6001
51 M 1902 76 IM
MCCARTHY, ROBERT P, 155 S 18TH,66102
342-7233
25 M 2834 53 U
MCCOWEN, HERBERT M, 3002 SENECA,66103
-
58 M 1902

MCCUNE, MARK A, 8919 PARALLEL PKWY,66112
492-6200
52 M 1902 77 D
MEBUST, WINSTON K, KU MED CENTER,66103
588-6148
33 M 5404 58 U
MEDHAT, MOHAMED A, KU MED CENTER REHAB MED,66103
588-6787
32 M 33002 54 RM
MEEK JR, JOSEPH C, KU MED CENTER,66103
588-6023
31 M 1902 57 IM
MESINA, ROLANDO R, 1102 MINNESOTA AVE,66102
371-3829
37 M 74801 61 GS
MEYER, MARK E, 2424 W 40TH,66103
677-4818
58 M 1902
MINER JR, PHILIP B, K U MED CENTER,66103
588-6990
46 M 702 71 GE
MOELLER, DONALD D, 155 S 18TH,66102
371-4301
34 M 1902 60 GE
MOORE, WAYNE V, K U MED CENTER,66103
588-6336
42 M 2604 70 PD
MORFFI, RAUL R, 1225 N 78TH,66112
287-8300
25 M 27501 51 IM
MULLEN JR, CLIFFORD J, 9201 PARALLEL,66112
334-4250
38 M 1902 64 OPH
MULLEN SR, CLIFFORD J, 1828 WASH BLVD,66102
-
98 M 3006 23 OD
MURPHY, JOHN F, 3838 RAINBOW #1106,66103
-
52 M 1902
NEFF, JAMES R, KU MED CENTER,66103
588-6198
40 M 1902 66 ORS
NEIGHBOR, ERNEST G, 1420 S 42ND,66106
-
06 M 1902 33 FP
NEIGHBOR, ERNEST H, 1420 SOUTH 42ND ST,66106
831-1100
40 M 1902 66 ORS
NEIGHBOR, GAYLORD P, 1420 SOUTH 42ND,66106
831-1100
13 M 1902 41 FP
NELSON, WILLIAM PAUL, KU MED CENTER,66103
588-6015
30 M 1002 56 CD
NEWKIRK, DARREL D, 2427 N 73RD,66109
321-4803
42 M 2101 67 PH
NIBBELINK, LARRY WAYNE, 155 S 18TH,66102
371-2330
48 M 2803 75 OBG
NOBLE, MARK J, KU MED CENTER-UROLOGY,66103
588-6148
49 M 1902 71 U
NOHE, PHILIP C, 266 NEW BROTHERHOOD BLDG,66101
321-4422
17 M 1902 42 ORS
NORRIS, CHARLEY W, KUMC,66103
588-6700
33 M 1902 64 OTO
NOTHNAGEL, ARNOLD F, 501 WESTVALE,66102
-
15 M 1902 39 OD
NUNEZ, JULIAN, 4531 ORVILLE #209,66102
299-2088
30 M 27501 60 FP
O'BOYNICK II, PAUL LEONARD, KU MED CENTER,66103
588-5000
48 M 1902 73 NS
OLOFIELD, RAY W, 4631 ORVILLE,66102
287-3537
45 M 1902 72 FP
OTHMER, EKKEHARD, K U MEDICAL CENTER,66103
588-6440
33 M 40721 66 P
OVERFIELD, A SCOTT, 3838 RAINBOW APT 1105,66103
-
57 M 1902
OXLER JR, JOHN EDWARD, 8901 W 74TH SUITE 208,66204
362-6161
46 M 1902 72 IM
PALMER, MARVIN M, 8919 PARALLEL PKW,66112
788-9797
45 M 702 71 OBG

PALMERI, MARIA, 4631 ORVILLE,66102
596-1185
49 F 13201 75 FP
PALMERI, V CARLOS, 4631 ORVILLE, 66102
596-1185
46 M 13201 75 FP
PARDO, MANUEL P, K U MEDICAL CENTER,66103
588-6464
35 M 74801 62 P
PAREKH, AJITKUMAR M, 6013 LEAVENWORTH RD,66104
299-2069
47 M 49501 71 PUD
PARK, CHAN H, K U MEDICAL CENTER,66103
588-6029
36 M 58302 62 IM
PARRA, MIGUEL O, 6013 LEAVENWORTH RD,66104
299-2088
37 M 84710 64 FP
PASCHINO, ANTONIO R, 4631 ORVILLE #215,66102
287-7100
22 M 13201 60 P
PAZELL, JOHN A, 4631 ORVILLE,66102
287-6464
40 M 2501 66 ORS
PECANA, MANUEL C, BETHANY MED CENTER,66102
281-8896
45 M 74801 69 ANES
PERRY JR, LAWRENCE L, KUMC 39TH AT RAINBOW,66103
588-6522
34 M 1902 59 FP
PETERS, GLENN R, 2211 N 88TH,66109
-
12 M 1902 37 OD
PIERCE, GEORGE E, KUMC - PO BOX 255,66103
588-6115
33 M 2307 60 TS
PISCHKE, FRANK J, 8800 W 75TH SUITE 115,66102
321-1161
36 M 1902 62 OTO
POL, P ALBERT, 6013 LEAVENWORTH RD,66104
287-7100
36 M 84710 67 P
PORTER, DAVID M, 4517 TROUP,66102
287-8800
39 M 4707 64 PD
POTTER, ROBERT L, 1969 N 33RD,66102
321-0341
38 M 1902 64 IM
POWERS, G ROBERT, 8919 PARALLEL PKWY,66112
299-1469
33 M 1902 65 FP
PREMSINGH, NALINI G, 4631 ORVILLE #202,66102
596-2000
39 F 49508 65 CD
PRESKORN, SHELDON H, K U MED CENTER,66103
588-6400
48 M 1902 74 P
PRESTON, DAVID F, KU MED CENTER,66103
588-6810
33 M 3941 59 NM
PRETZ, JAMES B, 1610 WASHINGTON BOULEVARD,66102
342-2442
24 M 1902 47 FP
PRICE, HILTON I, KU MED CENTER - RAD DEPT,66103
588-6831
49 M 83601 72 OR
PRICE, JAMES GORDON, K U MED CENTER,66103
588-6510
26 M 702 47 FP
PRIETO, JORGE N, 6013 LEAVENWORTH RD,66104
299-2069
45 M 26401 69 GS
PROCHASKA, MARK L, RAINBOW TOWERS APT 1105,66103
-
57 M 1902
PROUD, G ONEIL, KU MEDICAL CENTER,66103
588-6700
13 M 2802 39 OTO
PUGH, DAVID M, K U MED CENTER,66103
588-6015
29 M 801 58 CD
QUINN, CHARLES E, 4601 ORVILLE,66102
287-6604
43 M 4707 68 OBG
RAGLAND, CHARLES W, 155 S 18TH,66102
321-2974
50 M 1902 77 IM
RECKLING, FREDERICK W, KUMC,66103
588-6129
34 M 3545 59 ORS
REDDY, EASHWER K, K U MED CENTER - RAD DEPT,66103
588-7350
44 M 49597 68 TR

REDFORD, JOHN W B, K U MED CENTER,66103
588-6777
29 M 5501 53 PM
REES, RONALD JOSEPH, 155 S 18TH,66102
371-4343
46 M 3006 72 DR
REED, JAMES STEWART, K U MED CENTER,66103
588-6019
46 M 3601 72 GE
REGIER, HENRY L, 2000 WASHINGTON BOULEVARD,66102
81 M 1902 07 DD
REILLY, DOUGLAS K, KU MED CTR PSY DEPT,66103
46 M 3005 74
RHODES, JAMES B, KU MEDICAL CENTER,66103
588-6019
28 M 1902 58 GE
RHODES, MARTIN L, 155 S EIGHTEENTH,66102
321-0341
47 M 1902 72 IM
RICE JR, FREDERICK A, 1029 N 32ND ST,66102
281-5252
36 M 4812 63 ORS
RILEY, RAY B, 2020 ORVILLE,66102
05 M 1902 36 DD
RINKENBERGER, ROBERT, K U MED CENTER,66103
588-6015
44 M 1902 71 CD
RISING, JESSE D, KU MEDICAL CENTER,66103
588-1934
14 M 1902 38 IM
ROBINSON, DAVID W, KU MEDICAL CENTER,66103
588-6136
14 M 4101 38 PS
ROBINSON, JOHN D, K U MED CENTER,66103
588-6670
48 M 1902 74 ANES
ROBINSON, RALPH G, KU MEDICAL CENTER,66103
588-6810
37 M 1902 62 NM
RODRIGUEZ, RAUL G, 6013 LEAVENWORTH RD,66104
299-2525
42 M 25401 68 P
ROOK, LEE E, 4116 STRONG,66106
831-2834
09 M 1902 38 FP
ROSENTHAL, STANTON J, K U MED CENTER,66103
588-6800
46 M 1902 71 DR
ROTH, ALAN E, BETHANY HOSPITAL 51 N 12,66102
281-8814
35 M 1902 62 PATH
RUBIN JR, BEN, 132 S SEVENTEENTH,66102
371-2561
37 M 3005 61 PD
RUTH, WILLIAM E, K U MED CENTER,66103
588-6044
26 M 1902 53 PUD
RYAN, MICHAEL J, 764 NEW BROTHERHOOD BLDG,66101
342-7070
14 M 2834 37 DTD
SAVIN, VIRGINIA J, KU MED CENTER,66103
588-6983
44 F 4112 70 IM
SCHIMKE, R NEIL, KU MED CENTER,66103
588-6043
35 M 1902 62 IM
SCHOTLAND, EDWARD S, 1300 N 78TH STE 303,66112
334-5250
36 M 86901 66 D
SCHREPFER, ROSEMARY, KU MED CENTER,66103
588-6200
22 F 1902 47 DBG
SCHWEGLER, RAYMOND A, 8919 PARALLEL PKWY,66112
492-5200
37 M 1902 63 CD
SCHWORM, CURTIS P, 155 SOUTH 18TH,66102
371-4343
47 M 3005 73 DR
SERERES, EDGAR P, 662 NEW BROTHERHOOD BLDG,66101
321-2501
15 M 1902 39 FP
SHERIDAN, RANDY M, 3709 EATON,66103
53 M 1902
SHERMAN, ROBERT P, 9201 PARALLEL AVE,66112
334-0040
34 M 1902 63 PUD
SIFERS, EARL C, 155 S 18TH,66102
371-2900
24 M 1902 47 GS
SIFERS, TIMOTHY M, 155 S 18TH,66102
371-2900
48 M 1902 74 GS
SILVERS, ALVIN, 1702 SW BOULEVARD,66103
236-9691
18 M 1902 45 FP
SKIKNE, BARRY S, KU MED CENTER,66103
588-6031
45 M 83601 61 HEM
SMITH, STEPHEN D, K U MED CENTER,66103
588-6300
45 M 3006 71 PD
SNODGRASS, WAYNE R, KU MED CENTER PED DEPT,66103
588-5318
45 M 1720 68 PD
SOUCEK, CHARLES D, 155 S 18TH,66102
371-4343
31 M 3005 56 R
SPAKE, ROBERT V, KU MED CENTER ENT DEPT,66103
588-6700
52 M 1902 78 OTO
SPAULDING, JOHN S, KU MED CENTER,66103
588-5900
29 M 1502 59 PD
SPEER, LELAND, 1022 HOEL PARKWAY,66102
12 M 1902 36 OO
STECHSCHULTE, DANIEL J, K U MED CENTER,66103
588-6008
36 M 2834 62 A
STEELE, CLARENCE H, 255 BROTHERHOOD BLDG,66101
321-1161
14 M 1902 40 OTO
STEINZEIG, SHERMAN M, 155 S 18TH,66102
621-1151
25 M 1902 52 CD
STEPHENS, RONALD L, KUMC,66103
588-6029
39 M 1902 65 IM
STUBBLEFIELD, CHARLES T, 155 S 18TH,66102
371-2330
32 M 1902 58 OBG
TAYLOR, SARAH A, KU MED CENTER-STUDENT UN,66103
588-6029
50 F 1902 75 IM
TAYLOR, THOMAS L, 155 S 18TH ST,66102
371-2900
40 M 1902 66 GS
TEMPLETON, ARCH W, K U MEDICAL CENTER,66103
588-6805
32 M 3005 57 R
THEROU, LEONA F, K U MEDICAL CENTER,66103
588-5302
41 F 5701 67 PD
THOMAS, JAMES H, K U MED CENTER,66103
588-5901
41 M 2012 66 GS
THOMAS, THOMAS V, 211 INDIAN SPRINGS MED BL,66102
287-2600
37 M 49549 61 CDS
THOMPSON, DANNIE M, ONE GATEWAY CENTER,66101
321-3355
35 M 4707 64 OBG
TOSONE, STEVEN R, K U MED CENTER,66103
588-6670
52 M 1902 77 ANES
TRUEWORTHY, ROBERT C, KU MED CENTER,66103
588-5701
40 M 2802 66 PD
UTLEY, JAMES HARMON, 51 N 12TH,66102
281-8880
51 M 1606 74 EM
VAN THULLENAR, PHILIP A, BETHANY MED CENTER,66102
281-8834
31 M 2834 57 PATH
VARGHESE, GEORGE, K U MEDICAL CENTER,66103
588-6795
44 M 49509 69 PM
VATS, TRIBHAWAN S, KUMC,66103
588-5701
40 M 49529 63 PD
VELARDE, HUGO, 4601 ORVILLE AVE STE 14,66102
287-8400
M 64 GS
VISSER, VALYA E, KU MED CENTER,66103
588-6337
47 F 1803 73 PD
WADDELL, BILL D, 155 SOUTH 18TH,66102
321-0386
31 M 3901 56 IM
WALKER, JACK D, KU MED CENTER,66103
588-6522
22 M 1902 53 FP

WALKER, MAURICE A, 3214 STRONG AVE,66106
831-1433
04 M 1501 28 GS
WALTER, DONNAL C, KU MED CENTER,66103
588-6337
48 M 1902 76 PD
WALTERS, WILLIAM DAVID, 1428 S 32ND,66106
384-1630
50 M 1902 78 FP
WAXMAN, DAVID, K U MED CENTER,66103
588-1207
18 M 3515 50 IM
WEIGEL, JOHN W, UROLOGY DEPT KU MED CTR,66103
588-6148
29 M 1902 54 U
WELLER, ELIZABETH B, KU MED CENTER,66103
588-6464
49 F 60501 75 CHP
WELLER, RONALD ALAN, KU MED CENTER - PSY DEPT,66103
588-6464
48 M 2302 74 P
WILLIAMS JR, STERLING B, K U MEDICAL CENTER,66103
588-6200
41 M 401 73 OBG
WINTER JR, CALVERT J, 7845 WALKER,66112
371-2561
22 M 1902 47 PD
WISE III, JOSEPH EDWARD, 132 S 17TH,66102
371-2561
51 M 1902 76 PD
WISNER JR, JOHN HENRY, 229 S EIGHTH,66101
588-6431
46 M 1902 76 P
WOLF, KARL T, 621 NORTHRUP AVE,66101
-
14 M 1902 48 OO
WONG, BERT Y, K J MED CENTER,66103
588-6015
40 M 801 65 CD
WOOD, FRED M, KU MED CENTER,66103
588-6144
38 M 4706 62 ORS
WRIGHT III, ROBERT W, 4606 CAMBRIDGE,66103
588-6670
50 M 1902 79 ANES
WRIGHT JR, ROBERT W, 669 NEW BROTHERHOOD BLDG,66101
371-5344
24 M 1902 48 GS
YAKAR, DANIEL, KJ MED CTR RAD DEPT,66103
588-3600
32 M 55001 58 RT
ZAREMSKI, SHERMAN C, 4631 ORVILLE STE 209,66102
596-1185
33 M 1720 58 IM
ZIEGLER, DEWEY K, KU MED CENTER,66103
588-6985
20 M 2401 45 N
ZINN, THOMAS W, 155 S 18TH,66102
371-4343
41 M 1902 67 R

KANSAS CITY, MO—816

ALLEN, MARK LYNN, 3720 WYOMING SUITE 3,64111
-
53 M 1902
BOUDET, ROBERT A, V A HOSP,64128
861-4700
36 M 1103 62 GS
BROTHERS, MARY ELIZABETH, 2727 MAIN,64108
-
49 F 1902 74 GS
CALKINS, W GRAHAM, V A HOSP,64128
861-4700
26 M 2501 50 GE
CRONEMEYER, RICHARD L, 444 GLADSTONE BLVD,66124
-
52 M 1902
DAVIS JR, JAMES W, V A HOSP,64128
861-4700
33 M 4705 57 IM
DIEHL, ANTONI M, 618 MEDICAL PLAZA,64111
753-4414
24 M 2604 47 PDC
FALLON, JOHN H, 3730 WYOMING,64111
-
59 M 1902
FESTOFF, BARRY W, VA HOSPITAL,64128
861-4700
40 M 1102 77 N
GODFREY, ROBERT G, VA HOSP,64128
861-4700
27 M 1902 58 RHU
HAQUE, ANWAR U L, 7902 SYCAMORE #1,64138
588-1700
51 M 70402 76 PATH
HARD, BENJAMIN F, MOBAY CHEMICAL CORP,64120
242-2525
28 M 4802 55 DBG
HOHLY, EVE K, 3923 HARRISON,64110
-
40 F 1902
JOHNS JR, LEO E, VETERANS MED CENTER,64128
861-4700
22 M 2101 45 PUD
JOHNSON, FREDERICK E, C/O MARY K BLOTT,64112
-
92 M 2843 21 OO
KINPORTS JR, EDWARD B, 2400 PERSHING RD STE 561,64108
842-1950
46 M 3006 74 EM
KINPORTS SR, EDWARD B, 2520 GRAND AVE #309,64108
-
15 M 1602 42 EM
LEWIS JR, H DANIEL, VETERANS MEDICAL CENTER,64128
861-4700
32 M 5101 58 CD
LUETJE, CHARLES MARION, 2928 MAIN SUITE 105,64108
561-4423
41 M 2803 67 DTO
OWENS, RICHARD L, 911 MAIN,64105
842-1146
24 M 3006 51 OM
PARDO, LILLIAN G, 4440 BROADWAY,64111
588-5909
39 F 74807 62 PDN
PAYNE, J RALPH, 4460 ROCKHILL TERRACE,64110
334-2500
40 M 1902 62 EM
POONAWALA, HUSENI E, 4400 BROADWAY SUITE 202,64111
561-2025
33 M 49528 59 P
REDING, DOUGLAS J, 5 W 57TH TERR,64113
588-5000
54 M 1803 80 IM
SINGER, PHILIP A, V A HOSPITAL,64128
861-4700
42 M 3545 69 N
SLEEPER, CAROL A, 20 E FOURTEENTH,64142
-
33 F 1902 58 PATH
SMITH, WILLIAM P, 109 W 69TH,64113
588-6886
51 M 1902 77 R
STASS-ISERN, MERRILL, 1405 W SOTH TERR,64112
281-8880
50 M 84706 77 EM
STILLIE DO, G DONALD, 7609 LOCUST,64131
-
48 M 2878 78 RT
STOKES, ROBERT LEE, 924 W 34TH,64111
281-8882
43 M 4804 74 EM
VITALE, NEIL B, 1135 W 41ST,64111
-
55 M 1902
WIEGMANN, THOMAS B, VETERANS MED CENTER,64128
-
43 M 40902 69 NEP
WIENS, TIMOTHY K, 3539 GENESSE,64111
-
55 M 1902
WONDER II, DONALD K, 1011 1/2 W 38TH,64111
-
53 M 4802 79 GS

KINGMAN—316
(Pratt-Kingman Society)

BLODM, L THEIL, BOX 496,67068
-
32 M 1902 57 R
BOYER, ROBERT E, 760 AVENUE D WEST,67068
532-5145
36 M 1902 63 FP
BURKET JR, GEORGE E, SPRING LAKE RTE 1,67068
-
12 M 1902 37 OO

KINSLEY—316
(Edwards County Society)

ATWOOD, M DALE, PO BOX 269,67547
659-2114
19 M 1902 51 FP

MCKIM, W LYNN, 109 WEST 8TH,67547
659-2137
33 M 1902 59 FP
SCHNOEBELEN, RENE E, 807 EAST 4TH,67547
659-2141
16 M 3901 40 FP

KIOWA—316
(Tri-County Society)

CHRISTENSEN, MARION O, 802 DRUMM,67070
825-4121
25 M 3901 52 FP
RODRIGUEZ, ALBERTO, 425 CAMPBELL,67070
825-4039
25 M

LA CROSSE—913
(Central Kansas Society)

BHARGAVA, ASHOK KUMAR, BHARGAVA CLINIC,67548
222-2564
37 M 49547 64 FP
BHARGAVA, SHOBHANA, BHARGAVA CLINIC,67548
222-2564
38 F 49547 64 FP

LANSING—913
(Leavenworth County Society)

SNOW, DONALD L, HOLIDAY PLAZA,66043
727-3204
21 M 64901 54 OBG

LARNED—316
(Pawnee County Society)

BRENNER, WILLIAM R, 804 CARROLL,67550
285-3133
15 M 3006 39 FP
CRAM JR, OLE R, 722 MANN,67550
285-2141
18 M 1902 43 FP
DAVIS, DAVID H, 815 W 6TH,67550
-
04 M 1902 30 OO
EWING, THOMAS O, 804 CARROLL,67550
285-3133
22 M 1902 46 FP
KEIL, JAMES E, 528 W 12TH APT 4,67550
755-3151
44 M 1902 70 P
PARAMESH, JAYA, ROUTE 3 BOX 89 LSH,67550
285-6285
32 F 49508 55 GP
SHAH, MIAN, SHAH CLINIC,67550
285-3173
32 M 70403 58 GS
SHAH, NASREEN, SHAH CLINIC,67550
285-3173
39 F 70409 62 OBG
SHEPARD, LEROY W, 603 W 5TH,67550
-
04 M 3006 30 OO
SMITH, JOHN D, 804 CARROLL,67550
285-3133
22 M 3901 51 FP

LAWRENCE—913
(Douglas County Society)

ANDERSON, WINSTAN L, 3603 W 10TH ST,66044
-
09 M 1902 34 OO
AUCHARD, VIRGIL M, 2126 LOUISIANA,66044
-
89 M 1902 23 OO
BAILEY, WILLIAM A, PO BOX 127,66044
843-9125
40 M 1902 66 ORS
BELOT JR, MONTI L, LAWRENCE NATIONAL BK BLDG,66044
843-6340
13 M 1902 40 FP
BIERI, PETER V, 1112 W SIXTH ST,66044
841-5217
45 M 1902 71 ENT
BISHOP, ROONEY LEE, 346 MAINE,66044
842-7200
49 M 1902 75 IM
BITTENBENDER, LEE R, 930 IOWA,66044
842-7001
46 M 1902 72 O
BLAIR, T RICHARD, 404 MAINE STE 3,66044
842-3535
34 M 1902 60 IM
BOYDEN, MARY S, MEDICAL ARTS BLDG,66044
842-3778
14 F 2604 38 POA
BRANSON, VERNON L, 346 MAINE,66044
842-4477
17 M 1902 42 PO
BRUNER, STEVEN C, 500 ROCKLEDGE,66044
841-6540
46 M 1902 72 FP
BRUNFELDT, JOAN KRAUS, 404 MAIN,66044
842-3635
52 F 1902 77 IM
BUCK JR, HENRY W, 1112 W SIXTH,66044
841-9200
34 M 1902 60 OBG
CARNAHAN, ROBERT L, 1112 W SIXTH SUITE 112,66044
841-4310
42 M 1902 70 IM
CHEDIAK, ELIAS, 501 MISSOURI,66044
841-7430
39 M 84704 65 P
CLINTON, DALE L, 15 E SEVENTH SUITE 103,66044
841-5716
21 M 1902 54 GP
CULVER, WARREN T, 2500 W 6TH,66044
842-4178
20 M 3508 46 OPH
DAHL, DENNIS R, 2927 HARVARD RD,66044
843-4455
30 M 1902 61 FP
DOUGLAS, JOSEPH MAHLON, 930 IOWA,66044
841-3535
39 M 1902 65 P
DUNLAP, RICHARD L, MEDICAL ARTS CENTER,66044
842-4344
12 M 3005 37 ENT
FRIESEN, DALE, PO BOX 521,66044
842-7026
47 M 1902 74 ANES
GILLES, HELEN M, MED ARTS CENTER,66044
842-4477
22 F 1902 45 PO
GOODWIN, PHILLIP A, 500 ROCKLEDGE,66044
841-6540
28 M 1902 55 ANES
HAGGAN, MARGARET E, WATKINS MEM HOSP,66044
843-4455
F FP
HASSELLE III, JAMES E, 601 MISSOURI,66044
841-7430
35 M 4706 59 P
HATTON, DONALD W, 404 MAINE ST,66044
842-3635
42 M 1902 68 IM
HERMES, RICHARD L, THE MEDICAL ARTS CENTER,66044
843-0677
15 M 4112 39 OBG
HIEBERT, DAVID L, 1112 W SIXTH,66044
841-3211
35 M 1902 61 R
HIRD, WAYNE E, 2802 TRAIL RD,66044
842-4300
26 M 1902 50 TS
HORTON, BILL GATE, 346 MAIN,66044
841-1028
47 M 1902 74 OPH
HUGHES, ROBERT WALTER, MEDICAL ARTS BLDG,66044
843-1374
27 M 1902 54 FP
INGHAM JR, H LAIRD, 404 MAINE,66044
842-3635
45 M 3901 70 IM
JONES, H PENFIELD, MED ARTS CENTER,66044
-
06 M 2401 31 GS
JOSEPH, HOWARD F, DOCTORS BUILDING,66044
843-3981
26 M 1902 51 J
LEARNED, GEORGE R, 401 ARKANSAS,66044
843-5502
22 M 1902 55 GS
LESSENDEN, GLENN A, MED ARTS BLDG,66044
843-6233
24 M 1902 48 FP

LOVELAND, G CHARLES, 346 MAINE,66044
842-4477
47 M 1902 73 PD
MADSEN, GLENN L, 1112 W SIXTH,66044
841-3211
38 M 3005 65 R
MANAHAN, G EUGENE, MED ARTS CENTER,66044
842-0211
19 M 1902 44 GS
MITCHELL, ALEX C, 1626 W 20TH,66044
843-4739
18 M 1902 50 PH
MODDRELL, CAROL A, 404 MAINE,66044
843-3680
45 F 1902 71 PATH
MONCKTON, LAURANCE A, 1112 W SIXTH SUITE 204,66044
843-2010
48 M 1902 74 GS
NASH, ROBERT A, 404 MAINE,66044
841-5960
31 M 1902 55 P
NELSON, RICHARD O, 935 IOWA,66044
843-0921
11 M 1001 41 FP
OELSCHLAGER, RONALD D, 1112 W SIXTH,66044
841-3211
43 M 1902 69 R
OLSON, CARL E, 935 IOWA,66044
842-9911
17 M 1611 46 FP
ORCHARD, RICHARD A, 1112 W SIXTH SUITE 212,66044
841-2280
41 M 2802 68 OPH
OWEN, GARRY D, 1112 W SIXTH,66044
841-9200
37 M 1902 63 OBG
PEES JR, GERALD BOYD, 1112 W SIXTH SUITE 210,66044
843-5160
45 M 1902 71 IM
PRAEGER, MARK A, 1112 W SIXTH SUITE 204,66044
843-2010
42 M 1902 68 GS
PRICE JR, LAURANCE W, C/O LAWRENCE CLINICAL LAB,66044
843-3680
33 M 1902 59 PATH
REED, RALPH R, 404 MAINE,66044
842-3635
27 M 1902 53 IM
REESE, JOHN L, 346 MAINE,66044
842-6644
35 M 1902 61 GS
REITH, PAUL, XX107 WESTON SQ MEADOWBRO,66044
-
M
ROBERTS, RICHARD S, 308 MAINE,66044
843-6137
19 M 2802 44 GS
SANDERS, J ALAN, LAWRENCE MEMORIAL HOSP,66044
842-2083
29 M 1902 60 PATH
SCHNOSE, GREGORY D, 1112 W SIXTH SUITE 210,66044
843-5160
51 M 1902 76 IM
SCHROEDER, SYDNEY O, WATKINS MEMORIAL HOSP,66044
864-4045
18 M 1902 44 P
SCHWEGLER, RAYMOND A, MEDICAL ARTS CENTER,66044
843-4455
07 M 2504 31 OBG
STIVERS, MARK T, 404 MAINE,66044
842-2083
47 M 2803 73 PATH
THOMSEN, STEVEN T, 500 ROCKLEDGE RD,66044
841-6540
47 M 3005 72 FP
TUCKER, DONALD R, 1517 INDIAN WELLS CT,66044
354-5100
31 M 1902 57 IM
VERNON, MARY C, 500 ROCKLEDGE,66044
841-5540
52 F 1902 77 FM
WELL, MICHAEL A, 944 KENTUCKY,66044
843-3981
41 M 1606 67 U
WERTZBERGER, JOHN, PO BOX 127,66044
843-9125
36 M 1902 63 ORS
WERTZBERGER, KENNETH LYNN, 1112 W SIXTH BOX 127,66044
843-9125
47 M 1902 73 ORS
WILCOX SR, HOWARD L, MEDICAL ARTS CENTER,66044
843-0677
18 M 3520 44 OBG
WOLLMANN, MARTIN, 2615 ORCHARD LN,66044
843-4455
26 M 1902 57 IM

LEAVENWORTH—913
(Leavenworth County Society)

AL-BAGHAL, MOHAMMAD, 113 DELAWARE SUITE A,66048
651-2200
45 M 87501 72 U
ANDERSON JR, HERMAN L, 1817 SENECA,66048
682-8700
49 M 2405 77 IM
ANWAR, M ZIA, 109 DELAWARE,66048
651-2977
39 M 11501 67 IM
ASHKAR, ADNAN A, 109 DELAWARE SUITE F,66048
682-6818
42 M 52801 73 OBG
BARRY, DAVID R, 500 EISENHOWER RD,66048
727-6000
42 M 1902 68 FP
BENNETT, CHARLES A, 1009 S BROADWAY,66048
-
96 M 1902 25 OD
BUESING, OLIVER R, 1619 FOURTH AVE,66048
-
10 M 2501 32 GS
CENAC, MARK T, 500 EISENHOWER RD,66048
727-6000
27 M 401 49 GS
COMBS, G RALPH, CUSHING PAVILLION,66048
-
79 M 4101 02 OD
COMBS, PETER S, 213 DELAWARE,66048
682-0242
14 M 4101 41 IM
DE SOUZA, DERRICK J, 213 DELAWARE,66048
651-7555
43 M 49501 66 GS
DUYSAK, SAMI, 520 6TH AVE,66048
682-6661
22 M 90201 47 IM
GERBER, HARRY A, 605 N SIXTH,66048
-
96 M 5506 30 FP
GRAHAM, KENNETH L, 500 EISENHOWER RD,66048
727-6000
21 M 3840 45 GS
GRAHAM, THOMAS W, 500 EISENHOWER RD,66048
727-6000
26 M 3840 50 IM
GRISOLIA, ANDRES, 424 WALNUT ST,66048
682-5400
27 M 84708 50 ORS
HAMMEKE, JOHN C, 3601 S 4TH ST TRAFFICWAY,66048
682-5201
27 M 401 61 OPH
JOHLER, TERRY HARTWIG, 500 EISENHOWER RD,66048
727-6000
40 M 2802 67 FP
JOHNSON, PAUL D, 520 SIXTH AVE,66048
682-6661
36 M 1902 61 FP
KAVI, NAGESH G, PO BOX 1204,66048
682-2000
32 M 49509 58 IM
LIPOFF, JAY I, 410 S FOURTH,66048
682-6950
50 M 3519 75 IM
MCCOLLUM, WILLIAM B, 3601 S FOURTH,66048
682-6661
41 M 1902 66 TS
MCKEE, RICHARD S, 1210 S BROADWAY,66048
682-1023
05 M 1902 35 ANES
MERRITT, W HENRY, 520 SIXTH AVE,66048
682-6661
14 M 702 39 GS
MILLS, VERNON A, 520 SIXTH AVE,66048
682-6661
51 M 1902 77 PD
PARKER, ROBERT W, 500 EISENHOWER RD,66048
727-6000
45 M 1902 71 FP
PRAY, CLAUDIA M, 1406 S BROADWAY,66048
682-1404
52 F 1902 74 PD
RABE, MELVIN A, 520 SIXTH AVE,66048
682-6661
14 M 1902 37 GS

STRUTZ, WILLIAM C, 68 WESTWDDD DR,66048
682-8868
08 M 5606 43 R
TAMBURINI, MARIO, 2605 S 16TH,66048
682-8950
16 M 13201 49 R
VDORHEES, CARROLL D, 520 SIXTH AVE,66048
682-6661
23 M 1902 52 FP
VOORHEES, GDRODN S, 520 SIXTH AVE,66048
642-6661
12 M 1902 39 IM

LEBO—316
(Flint Hills Society)

HUNTER, KENNETH R, ,66856
256-2565
07 M 1902 39 FP

LENORA—913
(Northwest Kansas Society)

STEICHEN, EDWARD F, ,67645
05 M 1601 31 FP

LIBERAL—316
(Seward County Society)

ALLEN, RAY E, 2 PLAZA DR,67901
624-5691
37 M 1902 64 IM
CAEDD, CARMELITA D, PD BDX 1643,67901
624-1651
41 F 74801 63 R
CAMPIDN, WDDDROW M, 121 W THIRD,67901
624-2594
13 M 1902 39 IM
GRIMES, I RD55, 222 W 15TH,67901
624-1675
27 M 3901 54 TS
HARRIS, NORVAN D, 222 W 15TH,67901
624-3811
20 M 1902 44 D8G
HOLCOMB, WILLIAM M, 15 E 11TH,67901
624-2252
31 M 3901 56 G5
KDONS, JESS W, 1210 N WASHINGTDN,67901
624-3841
27 M 1902 57 DPH
NEVINS, RICHARD L, PO BOX 1824,67901
624-1841
47 M 3901 73 FP
PALMER JR, H C, 2 PLAZA DRIVE,67901
624-5691
36 M 1902 63 IM
PRDCHAZKA, DTTD F, BDX 1809,67901
12 M 1902 38 DD
RATHBUN, EDWIN D, 610 W 11TH,67901
624-1841
36 M 1902 62 FP
REESE, JACK D, 15 E 11TH,67901
624-6226
32 M 1902 57 FP
SEAGD, CHARLDTTE L, 1031 N KANSAS,67901
624-1625
35 F 1720 60 PD
WADE, THEDDDRE E, 318 N LINCOLN,67901
04 M 512 30 DD
ZAINALI, ASSADDLLAH, 601 LILAC DR,67901
624-1651
46 M S1701 72 R

LINDSBORG—913
(McPherson County Society)

FREDRICKSON, DUANE E, 121 W LINCDLN,67456
227-3371
39 M 1902 66 FP
FULLER, DERYL D, 121 W LINCOLN,67456
227-3371
25 M 1902 50 FP
MURFITT, MALCDLM C, 231 N MAIN,67456
227-2732
13 M 801 41 FP

LINWOOD—913
(Leavenworth County Society)

FUNK, EDWARD D, BOX 144 A-1,66052
04 M 1902 41 ANES

LYNDON—913
(Franklin County Society)

STOUT, NILES M, ,66451
828-4521
16 M 1902 50 FP

LYONS—316
(Rice County Society)

GRIMES, JAMES T, 1221 W NDBLE,67554
257-5124
27 M 1902 53 FP
SIEMENS, RICHARD A, 1221 W NOBLE,67554
257-5124
30 M 1902 59 FP
WOLF, CURTIS V, 1221 W NOBLE,67554
257-5124
37 M 1902 64 FP

MADISON—316
(Flint Hills Society)

BROWNING, WILLIAM R, *,66860
273-6753
44 M 1902 73 FP

MANHATTAN—913
(Riley County Society)

BAKER, RICHARD B, 1133 CDLLEGE,66502
537-4200
42 M 4113 68 ORS
BALL, RALPH G, 215 S DELAWARE,66502
03 M 1902 27 DD
BARLOW, JOHN M, 1133 CDLLEGE,66502
539-3504
45 M 1102 71 OTD
BASCDM, CHARLES H, C/D LAFENE STU HLTH CTR,66506
532-6544
31 M 1902 55 FP
BASCOM, GEORGE S, 1133 CDLLEGE,66502
539-6574
27 M 2401 52 G5
BDESE, KENNETH M, 1133 CDLLEGE AVE,66502
776-4744
25 M 1902 56 FP
BRDWN, ROBERT M, 1133 CDLLEGE,66502
537-4940
31 M 1902 63 FP
BURDICK, BRUCE M, 117 S FIFTH,66502
776-9411
25 M 512 53 P
CATHEY, RDBERT H, 1133 COLLEGE AVE,66502
537-4990
42 M 1902 68 D
CRANE, C HERBERT, 1133 COLLEGE,66502
537-9030
22 M 3520 46 PD
DURKEE, WILLIAM R, 1133 CDLLEGE AVE,66502
776-4744
23 M 1902 45 IM
FAIRCHILD, JDHN A, 756 CDLLEGE HTS CIR,66502
539-4041
14 M 3006 41 DD
FISCHER, REX R, 1133 CDLLEGE,66502
776-1400
34 M 3005 60 D8G
FISHER, RONALD M, N CENTRAL GUIDANCE CENTER,66502
234-9566
50 M 3305 76 P

FREEMAN, FRED A, MANHATTAN MED CENTER,66502
537-8710
42 M 1902 69 U
GARDNER, JAMES O, 1133 COLLEGE AVE,66502
537-4940
43 M 2834 71 1M
HANCOCK, DANIEL E, 1133 COLLEGE PO BOX 128,66502
539-5363
45 M 2803 PATH
HEASTY, ROBERT G, 2030 SCHEU DR,66502
-
11 M 3519 38 OO
HOSTETTER, PHILIP H, 821 POYNTZ,66502
537-2544
17 M 1902 42 FP
HUNTER JR, JAMES S, 1133 COLLEGE,66502
539-5501
18 M 2301 41 OBG
JUBELT, HILBERT P, 1133 COLLEGE,66502
537-9030
19 M 1611 43 PD
KALOOR, RICHARD H, PO BOX 128,66502
539-5363
40 M 2401 66 PATH
KEMPTHORNE, CHARLES R, 519 N 11TH,66502
-
03 M 5605 32 OO
KIRK, THOMAS E, 2312 ANDERSON,66502
776-0450
44 M 3005 71 OPH
KLINGLER JR, EUGENE A, 1133 COLLEGE,66502
539-5341
35 M 1902 62 GS
KLOBASA, CHARLES L, GUIDANCE CENTER,66502
539-5337
49 M 2803 75 CHP
LAFENE, BENJAMIN W, 1844 ANDERSON AVE,66502
-
01 M 3806 31 OO
LOWE, STANLEY W, 1133 COLLEGE AVE,66502
776-3451
32 M 1902 59 OPH
LYONS JR, FRANK C, 1133 COLLEGE AVE,66502
539-7641
44 M 3840 70 OR
MARSHALL, RONALD L, 1133 COLLEGE AVE,66502
539-5322
42 M 3005 67 OBG
MARTIN, DANIEL C, LAFENE HEALTH CENTER KSU,66506
532-6544
30 M 1902 58 1M
MCKNIGHT, DAVID E, 1133 COLLEGE AVE,66502
539-7641
32 M 1902 62 R
MCNEIL, ELBERT O, 1133 COLLEGE,66502
537-9030
22 M 702 48 PD
MEEK, PALMER F, 1133 COLLEGE,66502
537-2651
45 M 1902 71 1M
MILLER, ABRAHAM H, 1133 COLLEGE,66502
527-2651
29 M 4101 54 IM
MOSIER, MICHAEL L, 215 SOUTHWIND PL,66502
776-9761
52 M 1902 77 FP
MOSIER, STEVEN J, 215 SOUTHWIND PLACE,66502
776-9761
49 M 1902 74 FP
MOWRY, GERALD L, 1133 COLLEGE,66502
776-1400
26 M 1902 53 OBG
OLNEY, ROBERT O, 1133 COLLEGE AVE,66502
539-7555
27 M 3005 51 GS
PETERSON, JACK T, 1133 COLLEGE,66502
539-5363
25 M 1902 50 PATH
PHILIPP, JOSEPH THEODORE, 1115 WATERS,66502
537-7373
45 M 1902 67 OPH
PHILLIPS, STEPHEN B, LAFENE ST HEALTH CENTER,66506
532-6544
17 M 1902 45 1M
REITZ, LELAND C, 1133 COLLEGE,66502
537-2651
36 M 1902 63 1M
REITZ, ROGER P, 1133 COLLEGE,66502
537-2651
32 M 1902 59 1M
ROSE, GRAHAM C, 1133 COLLEGE,66502
537-9030
46 M 4706 70 PD

STONE, G REX, 1133 COLLEGE AVE,66502
539-7555
29 M 1902 54 GS
TAYLOR, BARBARA O, 1133 COLLEGE,66441
357-4940
50 F 1902 75 1M
TIEMANN, WILLIAM H, 1133 COLLEGE,66502
537-4940
42 M 3005 67 FP
TOUT, ROBERT C, LAFENE STUDENT HLTH CTR,66502
532-6544
27 M 4812 53 FP
VOLKMANN II, HARLEY W, 1133 COLLEGE AVE,66502
539-7641
47 M 1902 72 R
WHITE, THADDEUS H, 1735 ANDERSON AVE,66502
-
15 M 1902 42 OO

MANKATO—913
(Republic County Society)

KIMBALL, RICHARD R, 102 S CENTER,66956
378-3511
45 M 1001 72 FP
SCHLOTTERBACK, WILLIAM E, 517 N HIGH ST,66956
378-3511
31 M 1902 61 FP

MARION—316
(Marion County Society)

ENSEY, T CRANFORD, 504 S ROOSEVELT,66861
382-2182
20 M 4804 47 FP
GUERRA, TOMAS H, 122 S CEDAR,66861
382-2137
33 M 84704 65 1M
MARTIN, JEFFERY L, 537 S FREEBORN,66861
382-3722
50 M 1902 78 FP

MARYSVILLE—913
(Northeast Kansas Society)

ARGO, DONALD A, 808 N 19TH,66508
562-2303
36 M 3005 64 FP
LAWS, LEWIS R, 809 N 19TH,66508
562-2303
25 M 1902 54 FP

McLOUTH—913
(Jefferson County Society)

SNOOK, ROBERT RUFUS, ,66054
796-6116
11 M 1902 42 FP

McPHERSON—316
(McPherson County Society)

BILLINGS, THOMAS, 400 WEST 4TH,67460
241-5500
39 M 1902 66 FP
BRANDSTED, ERNEST C, 400 W 4TH,67460
241-1654
18 M 1606 44 OBG
COLLIER, WILLIAM J, 400 W 4TH,67460
241-1766
25 M 3605 49 TS
DYCK, ARTHUR H, 1020 N WALNUT,67460
241-0357
03 M 1902 28 FP
FERREE, RICHARD ALLAN, 400 W FOURTH,67460
241-7400
51 M 3006 76 FP
JOHNSON, J RICHARD, 400 W 4TH,67460
241-4293
28 M 1902 55 1M
QUAND JR, BIBIANO B, MEMORIAL HOSP,67460
241-2250
40 M 74801 63 U

PIERSON, WEIR, BOX 1028,67460
241-1445
17 M 1902 44 FP
PRICE, VAUGHAN C, PO BOX 451,67460
-
05 M 4706 29 GS
THOMAS, GREGORY MCQUEEN, 400 W FOURTH,67460
241-7400
47 M 1902 73 FP

MEADE—316
(Iroquois County Society)

OONAHOE, OORVAL H, PO BOX 488,67864
873-2181
32 M 5501 63 FP
HILL, RICHARD H, 234 EAST CARTHAGE,67864
873-2113
18 M 1902 44 FP

MEDICINE LODGE—316
(Tri-County Society)

HOFFER, JOHN G, 910 N WALNUT,67104
886-3222
13 M 1902 44 GS
STUCKY, DEAN E, 901 N WALNUT,67104
886-5653
33 M 1902 60 FP

MINNEAPOLIS—913
(Saline County Society)

BARKER, STEVEN E, 311 N MILL,67467
392-2144
51 M 1902 76 FP
WEDEL, KENNETH D, 311 N MILL ST,67467
392-2144
32 M 1902 60 FP
WEDEL, KERMIT G, 311 N MILL ST,67467
392-2144
32 M 1902 60 FP

MINNEOLA—316
(Iroquois County Society)

STEPHENS, CHARLES, MINNEOLA CLINIC,67865
885-4202
33 M 2803 58 FP

MONTEZUMA—316
(Ford County Society)

PHAM, TOAI KE, MONTEZUMA CLINIC,67867
845-2251
34 M 94101 62 GP

MORAN—316
(Allen County Society)

NEVITT, J RUSSELL, P O BOX 430,66755
-
99 M 3510 32 OO

MOUNDRIDGE—316
(McPherson County Society)

JANZEN, HERMAN F, RR 1 BOX 27KK,67107
-
91 M 1902 35 GP
KAUFMAN, WILLARD E, 115 N CHRISTIAN AVE,67107
345-6322
28 M 1902 53 FP
LOGANBILL, VARDEN J, 115 N CHRISTIAN AVE,67107
345-6322
26 M 1902 54 FP

MULVANE—316
(Sedgwick County Society)

COBB, LESLIE H, 102 E MAIN,67110
777-1441
17 M 4804 47 FP

NASHVILLE—316
(Pratt-Kingman Society)

WAYLAN, THORNTON L, ,67112
-
06 M 1902 35 OO

NEODESHA—316
(Southeast Kansas Society)

CHRONISTER, BERT, PO BOX 118,66757
325-2622
38 M 1902 64 FP
MOORHEAD JR, F ALLEN, 709 MAIN ST,66757
325-2200
39 M 1902 65 FP

NESS CITY—913
(Central Kansas Society)

PRAKALAPAKORN, DARANEE, 412 N TOPEKA,67560
798-2233
47 F 89101 69 PD
PRAKALAPAKORN, YANYONG, 412 N TOPEKA,67560
798-2233
43 M 89101 69 GS
WIENS, PETER K, BOX B,67560
-
24 M 1902 55 FP

NEWTON—316
(Harvey County Society)

ALLEN, FRANCES A, 1112 BOYD,67114
-
15 F 1902 43 OO
BATES, MICHAEL NICHOLS, 126 S PINE,67114
283-4153
50 M 1902 75 OBG
BENTON, JAY S, 301 MAIN,67114
283-3600
23 M 4804 49 OBG
CAMPBELL, FRANCES S, 1901 E FIRST,67114
283-2400
35 F 4101 61 P
CARPER, IVAN H, BOX 627,67114
283-2800
28 M 1902 59 GS
CARPER, OWEN E, BOX 627,67114
283-2800
37 M 1902 64 FP
CLAASSEN, MILTON A, 201 S PINE ST,67114
283-3600
32 M 1902 58 ORS
CRAIG, CHARLES C, BOX 627,67114
283-2800
45 M 1902 71 ORS
CRANSTON, STEPHEN D, BOX 627,67114
283-2800
45 M 1902 71 GS
DYCK, GEORGE, PRAIRIE VIEW INC,67114
283-2400
37 M 6201 64 P
ENNS, EUGENE K, 5 INDIAN LANE,67114
-
15 M 1902 40 OO
FENT, LEE S, 316 OAK,67114
283-0505
14 M 2834 43 GS
FRANSEN, HERBERT, BOX 848,67114
283-5040
32 M 6501 60 GS

FRANSEN, PAUL H, BOX 848,67114
283-5040
45 M 6501 71 FP
GLOVER, RICHARD M, BOX 627,67114
283-2800
21 M 1902 53 FP
GRABER, CHARLES, BOX 848,67114
283-5040
44 M 3006 75 GS
GRISWOLD, DALE G, BOX 627,67114
283-2800
27 M 1902 53 IM
GROVE, JOHN A, 407 W 16TH,67114
-
08 M 1606 37 ORS
HENDRICKSON, JON R, BOX 627,67114
283-2800
51 M 1902 73 PD
HENDRICKSON, KATHRYN D, BOX 627,67114
283-2800
52 F 1902 77 PD
HWA, EUGENE C, 500 MAIN,67114
283-1160
21 M 24216 47 R
IRWIN, RICHARD L, 218 S KANSAS AVE,67114
283-1400
48 M 1902 75 OPH
ISAAC, CHARLES A, BOX 627,67114
283-2800
25 M 1902 49 U
KLIEWER, VERNON L, PRAIRIE VIEW MHC,67114
283-2400
31 M 1606 57 CP
KUMAR, SURINDER, 201 S PINE,67114
935-2241
46 M 1902 69 OBG
LINDHOLM, GERALD R, BOX 627,67114
283-2800
51 M 1902 76 FP
MYERS, ROBERT W, RT 1,67114
-
11 M 1902 43 OD
NACHTIGALL, ANDREW, BETHEL CLINIC,67114
283-3600
28 M 1902 59 PD
O'TOOLE, JAMES K, PRAIRIE VIEW HOSPITAL,67114
283-2400
28 M 1643 54 P
OLSON, ERWIN T, BETHEL CLINIC,67114
283-3600
19 M 1902 47 PD
PREHEIM, DELBERT V, BOX 848,67114
283-5040
13 M 702 42 IM
PRENTISS, HAROLD, 1305 TERRACE DR,67114
283-9433
36 M 1720 62 R
QAMAR, YUSUF, BOX 627,67114
283-2800
38 M 70409 62 IM
RADOVANOV, RADMILA, BOX 364,67114
-
34 F 95702 60 R
RICH, ELDON S, BETHEL CLINIC,67114
-
16 M 1902 46 GYN
SCHMIDT, HERBERT R, CEDAR VILLAGE,67114
-
03 M 1902 34 OD
SILLS, CHARLES T, 1631 HILLCREST,67114
-
09 M 1902 37 OD
SIMMONS, ROBERT EARLE, BOX 848,67114
283-5040
49 M 1902 74 IM
TANDOC JR, VALENTIN T, BETHEL CLINIC,67114
283-3600
39 M 74809 62 U
TOMPKINS, CARL D, 316 OAK STREET,67114
283-1380
22 M 1902 51 FP
VAUGHAN, DONNA A, 201 S PINE,67114
283-3600
45 F 1902 71 IM
VOGT, VERNON W, BETHEL CLINIC,67114
283-3600
22 M 3005 53 FP
VON GRASEMSIRI, SUNAN, 1709 CYPRESS LN,67114
283-1160
41 M 89101 67 R
WEBER, ROY R, BOX 848,67114
283-5040
45 M 1902 73 IM
WHEELER, DWIGHT E, BETHEL CLINIC,67114
283-3600
50 M 2012 76 IM
WIENS, J WENDELL, 201 S PINE,67114
283-3600
32 M 1902 59 GS

NORTON—913
(Northwest Kansas Society)

COLIP, F MERLYNN, 711 N NORTON,67654
877-3305
35 M 1902 61 FP
COOPER, ARTHUR E, 305 W WILBERFORCE,67654
-
08 M 1511 34 GP
HARTLEY, ROY WILEY, 711 N NORTON,67654
877-3305
37 M 1902 63 FP
HARTMAN, ROGER L, 711 N NORTON,67654
877-3305
35 M 1902 61 FP
LONG, ROBERT C, 711 N NORTON,67654
927-3305
27 M 1902 53 GS

NORTONVILLE—913
(Jefferson County Society)

MADISON, WILLARD A, ,66060
886-2110
20 M 1902 51 FP

OAKLEY—913
(Northwest Kansas Society)

OHMART, RICHARD V, PO BOX 756,67748
672-3262
36 M 1902 62 FP
SEKAVEC, GORDON B, 209 CENTER AVE,67748
672-3351
07 M 1902 38 FP

OBERLIN—913
(Northwest Kansas Society)

PALMER, MARGJERITE L, BOX 110,67749
475-2221
25 F 5404 54 GP
SIMPSON, ROBERT LIMBAUGH, 902 W COLUMBIA,67749
475-2221
25 M 4706 51 GS
WHITAKER, REN R, 902 W COLUMBIA,67749
475-2222
37 M 5404 66 FP

OLATHE—913
(Johnson County Society)

ARONOFF, MICHAEL E, 407 S CLAIRBORNE STE 209,66062
782-3953
39 M 1604 64 ENT
BEEBE, EDMER, 420 EAST CEDAR,66061
-
03 M 5505 32 FP
BLISS, JOY V, 42 HOLLY DRIVE,66062
782-2292
42 F 3005 68 ANES
BLUM DO, MICHAEL A, 401 S CLAIRBORNE,66062
764-7060
47 M 2878 73 PD
BROWN, PAUL W, 405 S CLAIRBORNE,66062
782-3322
44 M 1902 70 FP
CHENOWETH, JOHN R, D.O., 1003 STRATFORD RD,66062
-
36 M 2878 70 OST
CLENDENIN, ROBERT KEELE, 1950 EAST SANTA FE,66062
764-6160
48 M 1902 73 EM
DELPHIA, ROBERT E, 401 S CLAIRBORNE,66062
782-1610
24 M 1902 56 FP

DUNN, MARTIN J, 407 S CLAIRBORNE STE 104,66062
764-1125
49 M 2803 76 O
EIOT, DAVID W, 407 S CLAIRBORNE,66062
782-8487
44 M 2501 70 FP
EIOT, LAURENCE A, 407 SOUTH CLAIRBORNE,66062
782-8487
44 M 1902 71 FP
FORTUNE, CEORIC B, 405 S CLAIRBORNE,66062
782-3322
40 M 1902 66 FP
GLAZZARO, CHARLES D, 407 S CLAIRBORNE,66062
782-3384
28 M 2507 56 P
HALVORSON, HOWARD C, 407 S CLAIRBORNE,66062
782-2020
41 M 5404 66 U
HUDSON, ROBERT P, 12925 FRONTIER RD,66061
588-7040
26 M 1902 52 IM
JENSEN, THOMAS M, 407 S CLAIRBORNE,66062
782-1148
47 M 3005 73 ORS
LAIRD, DALE D, ONE PATRONS PLAZA,66061
782-3631
42 M 1902 68 OPH
MATHEWS, ROBERT C, 300 S ROGERS RD,66062
782-1451
50 M 2834 77 EM
MATTHEW, WILLIAM L, 405 S CLAIRBORNE,66062
782-3322
29 M 1902 56 FP
MCCANN, WILLIAM E, 540 E SANTA FE,66061
782-0262
22 M 3901 48 FP
MEE, ADRIAN W, 28 HOLLY DRIVE,66062
782-2292
19 M 1902 54 ANES
MELENDRES, JUANITO M, 7400 GLEASON RD - RR 1,66061
684-4649
36 M 74809 61 PD
MENDLICK, R MICHAEL, 407 S CLAIRBORNE STE 101,66062
782-1148
44 M 1902 70 ORS
MILLIGAN, DONALD B, 401 S CLAIRBORNE,66062
782-1610
48 M 2307 74 FP
MORGAN II, DAVID LLOYD, 807 S CLAIRBONRE,66062
782-8300
49 M 2820 77 IM
PIERRON, GEORGE J, 540 EAST SANTA FE,66061
782-0260
22 M 1902 47 FP
PINCOMB, ARTHUR L, 401 S CLAIRBORNE,66062
782-1415
20 M 1902 51 FP
ROMONDO, STEVEN A, 300 SOUTH ROGERS RD,66062
782-2292
47 M 1902 73 ANES
RUHLEN, JAMES L, 807 S CLAIRBORNE,66062
782-8300
46 M 1902 72 IM
SEAMAN, LAUREN I, 1010 S CLAIRBORNE,66062
-
07 M 6001 38 FP
SETTLE JR, RUSSELL D, 407 S CLAIRBORNE,66062
782-3384
35 M 1902 60 P
SHANKER, STUART G, 401 S CLAIRBORNE STE 200,66062
764-7060
49 M 2803 75 PD
SHEFFER, KEITH D, 407 S CLAIRBORNE STE 101,66062
782-1148
37 M 1720 67 ORS
SNYDER JR, RICHARD HENRY, 300 S ROGERS ROAD,66061
782-1451
45 M 1902 73 ANES
STITES III, HAROLD W, 807 CLAIRBORNE,66062
782-8300
50 M 2803 80 IM
VILLANUEVA, CESAR L, 401 S CLAIRBORNE,66602
764-4467
39 M 74802 65 OBG
YEOMANS, RONALD N, 405 S CLAIRBORNE SUITE 4,66062
782-3073
40 M 1902 67 OBG

ONAGA—913
(Pottawatomie County Society)

FLECKENSTEIN, CHARLES S, 501 LUCIEN ST,66521
-
07 M 1902 36 OD

WALSH, THOMAS E, ONAGA CLINIC,66521
889-4241
48 M 1902 74 FP

OSAGE CITY—913
(Flint Hills Society)

ADAMS, DWIGHT, 603 HOLLIDAY,66523
528-3165
M 1902 GP
WILLIAMS, HOMER J, 611 S SIXTH,66523
-
05 M 1902 31 FP

OSAWATOMIE—913
(Miami County Society)

APPENFELLER, WILLIAM D, 524 BROWN AVE,66064
755-3166
25 M 1902 53 FP
MORALES, AMALIA D, P O BOX 500,66064
755-3151
22 F 21501 50 AOM
MORALES, OTTO E, P O BOX 500,66064
755-3151
22 M 27501 51 P

OSWEGO—316
(Labette County Society)

BURGESS, ARTHUR P, 504 5TH STREET,67356
795-4427
19 M 1902 52 FP
SHORTES, LOIS E, 408 E FIFTH,67356
795-4868
F FP

OTTAWA—913
(Franklin County Society)

BLANKENSHIP, JIM D, 1320 S ASH,66067
242-5581
50 M 2834 76 FP
CORDER, S SCOTT, 1502 S CEDAR,66067
242-2641
51 M 1902 76 FP
GOLLIER, ROBERT A, *,66067
-
13 M 1902 37 OD
GOLLIER II, ROBERT A, 1320 S ASH,66067
242-1520
40 M 1902 66 FP
HADLEY, DELMONT C, 1320 SOUTH ASH,66067
242-3891
35 M 1902 64 FP
HENNING, CALVIN W, 1502 CEDAR,66067
-
05 M 1902 35 FP
LAURY, DAVID G, 1320 S ASH,66067
242-1520
17 M 1606 44 FP
PHILGREEN, DONALD E, 1320 S ASH,66067
242-3891
39 M 1602 67 FP
RANSOM, WILLARD B, 1320 S ASH,66067
242-1620
49 M 1902 77 FP
REYES JR, FRANCISCO A, 1320 S ASH,66067
242-5312
38 M 74801 61 GS
SPEER, LOUIS N, PO BOX D,66067
242-1257
14 M 1606 41 FP
STREHLOW, CHESTER H, PROFESSIONAL PLAZA BLDG,66067
242-3891
30 M 1902 57 FP

OVERBROOK—913
(Shawnee County Society)

RUBLE JR, JAMES L, OVERBROOK COMM CLINIC,66524
665-2205
26 M 1902 53 FP

PAOLA—913
(Miami County Society)

BANKS, ROBERT E, PO BOX 298,66071
294-2305
29 M 1902 55 FP
NAIK, GOPAL V, 700 BAPTISTE DR,66071
294-5316
46 M 49501 71 OBG
ROWLETT, JACK G, PO DRAWER A,66071
294-2356
21 M 1902 52 FP
STANLEY, REX C, PO DRAWER A,66071
294-2355
24 M 1902 52 GS

PARSONS—316
(Labette County Society)

AVES, AGNES, 1509 MAIN,67357
421-0600
38 F 74801 59 IM
AVES, RENATD B, 1509 MAIN STREET,67357
421-0600
35 M 74801 59 GS
BOLT, MICHAEL, 400 KATY,67357
421-2700
51 M 1902 76 GS
CAREY, LARRY J, LABETTE CO MED CL SUITE 5,67357
421-8361
51 M 1902 74 FP
CRAMER, GUY W, 412 MURDOCK,67357
-
11 M 1902 39 DO
DAIZ, ANTONID S, LABETTE COUNTY MED CTR,67357
421-4880
37 M 74910 63 DR
DILLON, WILLIAM L, LABETTE CO MED CL BOX H,67357
421-0881
45 M 1902 71 ORS
HENDERSON, CHARLES F, 1617 GRAND,67357
-
14 M 1902 40 OO
HOMPLUEM, PACHAREE, LABETTE CO MED CL BOX 256,67357
421-2460
43 F 89102 69 PD
KISHORE, ROY N, 3604 GABRIEL APT 221,67357
421-4251
44 M 49511 66 OTO
KISHORE, SHEELA, 3604 GABRIEL APT 221,67357
421-2741
43 F 49511 66 ANES
LAVA, CHIRUND, PO BOX 290,67357
421-6210
40 M 89102 63 GS
MARTIN, EARL A, 1516 GRAND,67357
-
07 M 1606 35 OO
MILLER, CHARLES H, 2819 CLARK,67357
-
07 M 3006 32 OO
MILLER, DEAN M, 203 CRESTVIEW,67357
421-4880
22 M 1902 48 R
MILLER, STEPHEN FRANCIS, 1509 MAIN,67357
421-0600
45 M 1902 70 GS
PACE, JOHN D, KATY HOSPITAL CLINIC,67357
-
94 M 1902 20 FP
PAI, RADHA V, PO BOX 1057,67357
421-0080
45 F 6701 ANES
PAI, VARADARAJ S, PO BOX 1057,67357
421-0080
42 M 6701 U
PARANJOTHI, SUBRAMONIAM P, 1509 MAIN,67357
421-6160
39 M 49531 64 IM
PAULS, DANIEL N, PO BOX 1014,67357
421-1431
45 M 1902 71 IM
ROTHSTEIN, TERRY B, 220 NORTH 32ND,67357
421-5900
43 M 1606 69 OPH
SHARMA, ARUN L, 1509 MAIN,67357
421-0600
46 F 49503 69 FP
SWARTZ, WARREN E, 400 KATY AVE,67357
421-2700
25 M 1902 52 GS
TANGCHUPONG, SARDHD, PO BOX 1054,67357
421-2460
43 M 89102 69 OBG
VERMA, ASHA, 400 KATY,67357
421-9090
37 F 49530 63 PD
WHITE, JOHN P, PARSONS CLINIC,67357
421-0500
17 M 1902 42 FP

PHILLIPSBURG—913
(Central Kansas Society)

RICHARDS, JON F, 205 W STATE,67661
-
50 M 1902 75 FP
WURM, MICHAEL J, 250 W STATE,67661
543-5267
48 M 1902 73 FP

PITTSBURG—316
(Crawford County Society)

ARMSTRONG, HAROLD J, PROFESSIONAL BUILDING,66762
232-2600
40 M 1902 68 ORS
BENA, JAMES H, 109 EAST 9TH,66762
231-6950
12 M 3005 36 PD
BERKEY, VERNON A, KIRKWOOD BLDG,66762
231-7650
18 M 1902 43 R
BIERLEIN, KENNETH J, 812 S CATALPA,66762
-
06 M 1606 33 OO
COOMER, TYLER E, 315 NATL BANK BLDG,66762
231-7730
30 M 2101 59 GS
COOPER, KENT J, 909 CENTENNIAL,66762
231-6280
41 M 1902 73 FP
ERICKSON, CLARENCE W, 217 NATL BANK BUILDING,66762
231-7400
06 M 1902 33 IM
ESCH, JOHN G, 207 KIRKWOOD BLDG,66762
231-5360
24 M 3006 48 GS
FREEMAN, MALCOLM C, 1102 VILLAGE DR,66762
231-6100
44 M 35207 68 ANES
GOMEZ, MODESTO S, 909 E CENTENNIAL #6,66762
231-2490
35 M 72601 63 PD
HOLSINGER, DONALD M, 1015 MT CARMEL PL,66762
231-5900
38 M 1902 64 IM
HUEBNER, ROBERT STEPHAN, NATIONAL BANK BLDG,66762
231-6160
42 M 1606 67 GS
HUERTER, DAVID F, 909 CENTENNIAL,66762
231-1650
46 M 1902 72 IM
LANCE, RAYMOND W, 608 W QUINCY,66762
-
22 M 1902 47 OD
LEFFLER, PAUL B, 309 WINWOOD,66762
-
02 M 1902 40 OO
MILLER, EARL E, 1312 S BROADWAY,66762
231-6410
13 M 1902 37 ENT
MULLER, SAMUEL B, 611 W QUINCY,66762
-
05 M 1902 34 OO
NEWMAN, CLIFFORD B, 1204 E 7TH,66762
-
01 M 1902 28 OO
ODGERS, RODNEY K, 909 CENTENNIAL,66762
231-4300
M 1902 74 IM
PAPP JR, S DEAN, R 5 BOX 293,66762
231-7650
46 M 1902 72 DR
PARSI, MANUTCHEHR K, 909 CENTENNIAL,66762
231-3770
38 M 51701 64 OBG
POGSON, GEORGE W, 1015 MT CARMEL PLACE,66762
231-5900
24 M 1902 47 IM

RAMIREZ, AUGUSTO H, 909 CENTENNIAL,66762
231-1600
32 M 26407 58 IM
RAMIREZ, IRENE, 909 CENTENNIAL,66762
231-6280
F PD
SCHLEMMER, ROGER B, 1009 S BROADWAY,66762
231-6380
37 M 1902 68 OPH
SEGLIE, FLOYD RONALD, 909 CENTENNIAL DR SUITE 3,66762
231-6280
43 M 1902 69 FP
TWEET, FREDRICK A, MT CARMEL HOSPITAL,66762
231-6100
39 M 1602 66 PATH
WOOD, DOUGLAS H, 413 W JEFFERSON,66762
-
11 M 5505 36 OO
YAGHMOUR, TALAAT E, 905 NORTH LOCUST,66762
231-0850
40 M 33002 64 U
ZABEL, KENNETH P, 909 CENTENNIAL,66762
231-1650
37 M 1902 65 IM

PLAINVILLE—913
(Central Kansas Society)

PAGE, O VALE, 409 S COCHRAN ST,67663
434-4609
20 M 1902 51 FP
PEDERSON, ARNOLD M, 409 S COCHRAN,67663
434-4609
22 M 1902 51 FP

PLEASANTON—913
(Bourbon County Society)

JUSTUS, WILLIAM J, ,66075
352-6134
29 M 1902 55 FP

PRATT—316
(Pratt-Kingman Society)

AMBLER, CARL D, 200 COMMODRE,67124
672-6476
31 M 1902 57 R
BARKER, PATRICK N, 420 COUNTRY CLUB RD,67124
672-7411
45 M 1902 71 GS
BLACK, CYRIL V, 223 E 4TH,67124
672-6403
05 M 4802 30 GS
CHAFFIN JR, GOODLOE S, 420 COUNTRY CLUB RD,67124
672-7411
50 M 4705 75 FP
FILLEY, VERNON W, 310 E 2ND,67124
672-5555
13 M 3005 43 GS
FREEMAN, F GILES, 310 E 2ND,67124
572-5555
18 M 1902 44 FP
PITMAN, WILL D, 717 WEST 3RD,67124
-
98 M 1902 25 DO
QUENZER, RONALD W, 420 COUNTRY CLUB RD,67124
672-7411
46 M 1601 73 IM
SIBALA, JUSTO L, 200 COMMODORE,67124
672-7159
20 M 74802 49 R
SUITER, DANIEL JAY, 420 COUNTRY CLUB,67124
672-7411
44 M 1902 71 GE
THOMAS, R CULLEN, 420 COUNTRY CLUB RD,67124
672-7411
47 M 1902 72 GS
THORPE, FRANCIS A, 310 E 2ND,67124
672-5555
08 M 1506 35 FP
WOLFF, FREDERICK P, 223 E 4TH,67124
672-6403
20 M 1902 44 IM

PROTECTION—316
(Iroquois County Society)

GLENN, LYLE G, 146 BROADWAY BOX 447,67127
622-4686
12 M 1606 40 FP

QUINTER—913
(Northwest Kansas Society)

GUNTER, CARL C, QUINTER CLINIC BLDG,67752
754-3333
20 M 1902 51 FP
HIESTERMAN, HERMAN W, QUINTER CLINIC BLDG,67752
754-3333
23 M 1902 51 FP

RANSOM—913
(Central Kansas Society)

MCLAIN, KENNETH, BOX 247,67572
731-2295
21 M 1902 46 FP

RUSSELL—913
(Central Kansas Society)

ELDREDGE, LOUIS O, 222 S KANSAS,67665
483-6000
48 M 74809 79 GP
MERKEL, EARL D, SHIELDS BLDG,67665
483-2178
32 M 1902 57 FP
PANICHABHONGSE, SAMBHUNOH, 213 WEST 7TH,67665
483-2178
43 M 89101 67 GS
PETTIJOHN, WALTER J, 624 W 12TH,67665
-
12 M 1902 37 OO
STARKEY, JERALD L, 326 MAIN,67665
483-2178
30 M 1902 56 FP
SWANN, CLAIR L, 112 W 7TH,67665
483-4212
13 M 1902 39 IM
WHITE, FAGAN N, 356 W 5TH,67665
-
11 M 702 36 OO

SABETHA—913
(Northeast Kansas Society)

KENNALLY, KEVIN P, 1018 MAIN,66534
284-2141
53 M 1902 78 FP
MONTGOMERY, THOMAS ALLEN, 1013 WYOMING,66534
-
10 M 1902 49 OO
WENGER, GREGG D, 1018 MAIN,66534
284-2141
M
YULICH, JOHN O, PO BOX 227,66534
284-2125
33 M 1902 59 FP

SALINA—913
(Saline County Soceity)

ALLEN, MONTE L, 600 S SANTA FE,67401
827-0307
36 M 1902 61 OTO
ANDERSON, JODY, 737 E CRAWFORD,67401
827-7261
32 F 1902 59 IM
BAXTER, W REESE, P O BOX 1707,67401
825-8221
47 M 1902 73 FP
BROWN, ROBERT WAYNE, PO BOX 1747,67401
825-7251
23 M 1902 55 IM
BRUMMETT, RICHARD R, P O BOX 1707,67401
825-8221
34 M 1902 64 FP
BRUNGARDT, BERNARD A, 400 E BELOIT,67401
827-4433
21 M 3006 46 ANES
CATHCART-RAKE, WILLIAM F, BOX 360,67401
827-7261
48 M 1902 74 IM
CLARK, DAVID H, 617 E ELM,67401
825-8221
36 M 1902 62 FP

COFFEY, ROY B, 671 ELMORE DRIVE,67401
823-6397
24 M 1902 47 ORS
CONNELLY, MAURICE R, RR 3 BOX 44,67401
-
12 M 2002 38 GS
CONNER, BRIAN, 1S18 B EAST IRON,67401
825-2272
46 M 1902 OPH
COSSETTE, JERROLD E, 909 E WAYNE,67401
823-7225
46 M 1902 75 ENT
COVERT, THOMAS J, PO BOX 360,67401
827-7261
45 M 1902 71 PD
CULTRON, FRANK T, 800 E CRAWFORD,67401
823-8151
10 M 1643 38 OPH
D'SOUZA, BISMARCK C, 116-A S 7TH,67401
827-9526
45 M 49S01 67 R
DICKERSON II, W JOHN, PO BOX 918,67401
827-9631
51 M 300S 76 IM
DOWELL, JAMES C, 645 E IRON,67401
827-72SS
26 M 1611 49 IM
DRAEMEL, H RICHARD, 600 S SANTA FE ST,67401
827-0307
18 M 1902 53 DTO
DREHER, HENRY S, 737 E CRAWFORD,67401
827-7261
18 M 1902 43 IM
EATON, GLEN E, RR 6 BOX 3S9,67401
827-3064
28 M 1902 54 ANES
EATON, LESLIE F, RR 1 BOX 75,67401
-
06 M 1902 32 OO
ELLISON, PAUL D, 1499 E IRON,67401
825-7271
3S M 210S 60 OPH
FORSTER JR, LOUIS G, 2033 RAYMOND,67401
825-8221
47 M 1902 73 FP
FREEMAN, RAYMOND S, 737 E CRAWFORD,67401
827-7261
20 M 702 50 PD
GANS, FREDERICK A, 950 S ELEVENTH,67401
825-0366
22 M 2834 46 PD
GRIFFITH, FRANK H, 1493 E IRON,67401
827-0489
45 M 4813 75 OPH
GUNN, MARVIN R, 116A S 7TH PO BOX 1059,67401
827-9525
28 M 3901 54 R
GUZMAN, MANUEL, CKMHC,67401
823-6322
27 M 64901 54 P
HARBIN, GARY LYNN, 523 S SANTA FE,67401
823-7213
50 M 1902 75 DRS
HARRIS, NORMAN R, 430 S 7TH,67401
825-8191
30 M 1902 59 OBG
HASSLER, RANDY D, 645 E IRON,67401
827-9635
45 M 1902 U
HATTON, LLOYD W, 709 HIGHLAND,67401
-
06 M 1902 33 P
HESSE, FREDERICK J, PO BOX #918,67401
827-9631
50 M 1902 75 IM
HODGES, MERLE A, 430 S 7TH ST,67401
825-8191
34 M 1902 58 OBG
HOLMAN, JON B, PO BOX 61,67401
827-9366
33 M 1902 63 P
JACKSON JR, DELMAS A, 645 E IRON,67401
827-7255
35 M 2101 60 IM
KREHBIEL, MARK A, 617 E ELM,67401
825-8221
49 M 1902 74 FP
KRUCKEMYER, ALAN L, 645 E IRON,67401
823-2215
45 M 1103 71 ORS
LAKE, MAX S, 600 S SANTA FE,67401
827-0307
19 M 300S 43 DPH

LASLEY, DAVID A, 645 E IRON,67401
827-9635
22 M 1606 47 U
LIVINGSTON, CHARLES E, 400 E IRON,67401
827-9631
32 M 1611 57 GS
LUNGSTRUM, JACK E, PO BOX 1346,67401
823-2215
21 M 1902 59 DRS
MACY, NORMAN E, BOX 128S,67401
827-4053
35 M 1902 60 PATH
MACY, TED L, PO BOX 360,67401
827-7261
43 M 1902 71 GS
MARCHBANKS, DONALD L, 520 COUNTRY CLUB RD,67401
823-2380
24 M 1902 51 FP
MARSHALL, GEORGE W, PO BOX 1705,67401
225-3191
44 M 1902 70 OBG
MARTIN, OLIVER L, 715 E REPUBLIC,67401
827-9631
08 M 1902 37 DBG
MATHIS, JERRY L, 1112 ALBERT,67401
827-9343
35 M 1902 62 PDA
MAXWELL, GORDON E, PO BOX 918,67401
827-9631
29 M 1902 55 DBG
MCCRAE, SPENCER C, 519 S SANTA FE,67401
827-4424
18 M 3S09 43 ORS
MILLER, ELDEN V, 1928 RIDGELEA,67401
827-3061
19 M 1902 44 ANES
MITCHELL, JOHN C, 644 S OHIO,67401
827-3061
13 M 1902 38 GS
MOWERY, WILLIAM E, 737 E CRAWFORD,67401
823-6455
23 M 1902 47 GS
NICKELL, WAITSTILL B, 400 E IRON,67401
827-9618
24 F 1606 50 ANES
NICKELL, WENDELL K, 400 E IRON,67401
827-9618
26 M 1606 50 TS
NULL, WILLIAM G, PO BOX 918,67401
827-9631
31 M 102 57 PD
OBANDO, GUILLERMO, 2110 KNOLLCREST DR,67401
823-6219
3S M 26404 62 R
PALMER, GERALD K, PO BOX 128S,67401
823-7201
24 M 1803 53 PATH
RASSA, REZA P, 204 SEITZ DRIVE,67401
827-4411
33 M S1701 59 R
REECE, RICHARD J, 116A S 7TH,67401
827-9526
23 M 1902 49 R
RODERICK, JAMES E, 645 E IRON,67401
827-9635
23 M 1902 47 U
ROMEISER, REX S, 645 E IRON,67401
827-9635
41 M 1902 67 U
RUEB, ANDREW E, 11 CRESTVIEW DR,67401
827-6591
11 M 1506 3S GS
SCHMIDT, RAMON WARNER, 400 E IRON,67401
827-9618
39 M 1902 6S GS
SCOTT, CHESTER E, S19 S SANTA FE,67401
827-S549
23 M 1902 51 FP
SEBREE, STEVEN G, PO BOX 360,67401
827-7261
47 M 1902 73 DBG
SIMPSON, J COLBERT, 308 WEST SOUTH,67401
-
07 M 3006 38 OO
SLOO, MILO G, PO BOX 1346,67401
823-2215
41 M 1902 67 DRS
SMITH, BOYD E, BOX 128S,67401
827-4053
46 M 3005 72 PATH
SMITH, DAVID E, 2106 APPLEWOOD,67401
588-6100
50 M 1902 76 GS

SMITH, HAROLD R, PO BOX 360,67401
827-7261
19 M 1902 51 GS
STOSKOPF, LAWRENCE E, 2413 EDGEHILL,67401
685-4389
39 M 1902 72 ANES
STUEWE, BRAD R, PO BOX 360,67401
827-7261
49 M 1902 74 IM
TAYLOR, THOMAS F, 430 S OHIO,67401
827-0345
26 M 1902 53 FP
WAGENBLAST, HOWARD R, 737 E CRAWFORD,67401
827-7261
21 M 1902 49 FP
WATERS, CLARENCE N, 530 S 5TH,67401
823-6497
13 M 2834 48 O
WEBER, ROBERT W, 645 E IRON,67401
827-7255
25 M 1902 49 IM
WEBER II, RALPH H, PO BOX 360,67401
827-7261
44 M 3005 75 PO
WOERNER, DAVID R, 539 S SANTA FE,67401
825-9397
49 M 64914 75 FP

SATANTA—316
(Southwest Kansas Society)

JABEL, JUVENAL T, SATANTA CLINIC,67870
649-2771
43 M 74809 IM
TADURAN, VIRGILIO, SATANTA CLINIC,67870
649-2771
43 M 74810 43 PATH

SCOTT CITY—316
(Southwest Kansas Society)

DUNN, DANIEL R, 1210 RUSSELL,67871
462-3332
49 M 1902 26 FP
FIELDS, GALEN W, 1106 COLLEGE,67871
-
IS M 1902 49 OO
HOPKINS JR, B MORRISON, 804 CRESCENT,67871
872-2187
23 M 1902 53 FP

SEDAN—316
(Southeast Kansas Society)

LIM, CARLO, 104 W MAIN,67361
725-3171
44 M 74801 70 IM
TAYLOR, ELMER W, 120 WEST OSAGE,67361
725-3141
28 M 512 57 FP
WALKER, WILLIAM K, 111 E CHEROKEE,67361
725-3171
18 M 1902 45 FP

SENECA—913
(Northeast Kansas Society)

BERKLEY, NORMAN W, 15 SOUTH 5TH ST,66538
336-2128
31 M 1902 63 FP
GILBERT, J HOWARD, 522 MAIN,66538
-
05 M 1902 41 OO
LUEGER DO, JAMES JOHN, 201 N SIXTH,66538
336-6113
51 M 2878 78 GP
MC GEENEY, TERRY L, 201 N SIXTH,66538
336-6113
51 M 1902 77 FP

SHARON SPRINGS—913
(Northwest Kansas Society)

CHUNG, JOHN J, WALLACE CO MED CLINIC,67758
852-4214
23 M 58301 48 FP

SHAWNEE MISSION—913
(Johnson County Society)

ALLEN, MAX S, 5103 W 96TH TERR,66207
588-6988
11 M 1902 37 IM
ALLEN JR, WILLIAM R, 6734 GRANADA RD,66208
-
46 M 1902
ALTENBERND, ELVIN CONRAD, 7319 W 81ST,66204
648-2010
26 M 1902 54 FP
ALVAREZ, LUIS A, 10211 MISSION RD,66208
649-3400
32 M 64914 61 FP
ARMBRUSTER, ALBERT A, 9119 WEST 74TH SUITE 202,66204
362-9220
17 M 512 55 GS
ATHON, MERRILL D, 8501 DELMAR LANE,66207
642-4242
24 M 1902 54 FP
BADEEN II, LOUIS JOHN, 10550 QUIVIRA RD,66215
492-3344
49 M 2820 74 OPH
BAEKE, JOHN D, 6806 WEST 83RD,66204
642-4242
19 M 1902 52 FP
BAKER, WILLIAM STEVEN, 7700 W 63RD,66202
262-1843
47 M 702 73 P
BALANDFF, ARNOLD Z, 4601 W 109TH SUITE 122,66211
642-4040
42 M 1803 67 PD
BANSAL, SATISH C, 8901 W 74TH SUITE 147,66204
384-2220
38 M 49541 61 DRS
BAPTIST, JEREMY E, 5811 OUTLOOK,66202
432-0625
40 M 2846 78 A
BARE II, CHARLES F, 8901 W 74TH SUITE 353,66204
677-2460
43 M 1902 69 U
BARKER, ELIZABETH B, 4121 WEST 83RD SUITE 123,66208
381-6669
30 F 4706 55 P
BARKER, JAMES BERTON, 8901 W 74TH,66204
362-6310
31 M 4706 55 OTO
BARNETT JR, THOMAS E, 10550 QUIVIRA SUITE 290,66215
492-2555
52 M 1902 75 IM
BARNHART, RONALD J, 9119 WEST 74TH,66204
831-2334
41 M 2501 68 OBG
BARNHORST, DONALD A, 10550 QUIVIRA STE 510,66215
492-6200
37 M 2834 63 CDS
BARR, RICHARD N, 7301 MISSION ROAD,66208
432-4366
32 M 1902 57 DPH
BARRICK, BRUCE, SH MISSION MEDICAL CENTER,66201
676-2340
39 M 1902 65 PATH
BATTY, LARRY H, 9119 W 74TH,66204
831-2334
51 M 1902 77 OBG
BATTY, THOMAS V, 5555 W 58TH,66202
432-2080
21 M 3806 54 FP
BAUER, MARTIN L, 2700 W 51ST,66205
281-8881
47 M 1902 73 PD
BAXTER, KIRKMAN G, 5013 WOODSON,66202
-
57 M 1902
BEAMON, RICHARD F, 10500 QUIVIRA,66215
492-1000
47 M 2803 73 EM
BELL, DELORIS W, 4601 W 109TH SUITE 116,66211
341-6550
42 F 1902 68 DPH
BELZER, EDWARD G, PO BOX 7426,66207
381-8282
36 M 3005 58 PD
BERRY, JOHN M, 8800 W 75TH SUITE 320,66204
262-3288
47 M 1902 74 O
BIKALES, VICTOR WILLIAM, 7301 MISSION RD,66208
384-1311
13 M 2105 38 P
BISHOP, FRANCIS E, 7501 MISSION ROAD,66208
648-3533
20 M 1902 45 P

BLETZ, DONALD B, 10550 QUIVIRA SUITE 510,66215
492-6200
28 M 5104 58 IM
BOLES, J MICHAEL, 5949 NIEMAN ROAD,66203
631-1300
35 M 1902 61 FP
BOSWELL, H CRAIG, 10550 QUIVIRA STE 510,66215
492-6200
47 M 1803 73 ANES
BRAVERMAN, DAVID ELLIOTT, 4601 W 109,66211
341-1101
47 M 2507 72 PDO
BRIGGENS, JAMES G, 5H MISSION MEDICAL CENTER,66201
676-2340
22 M 1902 47 PATH
BROUCEK, FRANCIS J, 4121 W 83RD,66208
642-5845
32 M 1543 58 P
BROWN, WILLIAM R, 7301 MISSION RD,66208
236-8865
23 M 1902 48 IM
BROXTERMAN, STEVEN JOSEPH, 8901 W 74TH SUITE 225,66204
362-5510
51 M 1902 76 FP
BRUNING, ROGER MARION, 7301 MISSION SUITE 342,66208
384-0745
48 M 1902 70 FP
BUCKMAN, MARTIN SPALDING, 10550 QUIVIRA SUITE 290,66215
492-6200
49 M 2802 76 IM
BURGER, PAUL B, 5638 NIEMAN RD,66203
631-6114
25 M 2834 50 FP
BURTON, JERALD ALBERT, 10550 QUIVIRA,66215
492-6200
42 M 2846 73 IM
BUTIN, RICHARD E, 2000 W 48TH TERR,66205
—
54 M 1902
CAIN, IVAN W, 4200 SOMERSET STE 224,66208
649-4599
18 M 1902 44 DRS
CALKINS, JOHN W, 5213 ROSEWOOD DR,66205
—
51 M 1902 76 OBG
CALKINS, LARRY L, JOHNSON CO NATL BK BLDG,66207
649-0110
18 M 1902 43 OPH
CARDUFF, JAY J, 6300 GLENWOOD,66202
432-4480
25 M 3006 54 FP
CARRASCO, LENDR C, 8971 W 75TH,66204
341-2000
41 F 74801 63 PD
CARRO, TONY L, 5442 SKYLINE DR,66205
—
57 M 1902
CASTEEL, CHARLES K, 8901 W 74TH SUITE 357,66204
831-1003
34 M 3901 59 U
CATTANEO, ERNEST A, 6100 MARTWAY,66202
262-3930
39 M 1902 65 IM
CAVITT, ROBERT F, 9119 WEST 74TH,66204
831-0700
24 M 1902 48 GS
CEDERLIND, CRANSTON JAY, 8901 W 74TH SUITE 328,66204
236-6455
45 M 1902 71 OBG
CHANG, SHU FANG, 10200 W 75TH,66204
432-7885
25 F 24239 49 P
CHRISTIAN, STANLEY J, 9129 DELMAR,66207
—
18 M 1902 44 DD
CLYMER, DAVID V, 2309 W 71ST TERR, 66208
—
53 M 4705 80 ORS
COE, RICHARD O, 7301 MISSION RD SUITE 247,66208
362-8505
31 M 4804 56 OPH
COHEN, ROBERT A, 3700 W 83RD,66208
642-2100
39 M 2803 64 PD
COHN, STEVEN G, 8627 LINDEN DR,66207
334-2500
41 M 1902 67 ANES
COOLEY, DAVID A, 8800 W 75TH SUITE 300,66204
362-1225
40 M 1902 62 RHU
COOPER, JACK R, 7301 MISSION RD SUITE 330,66208
432-6300
17 M 3940 43 NS
COOPER, LEO F, 3818 W 57TH TERR,66205
588-6766
15 M 1902 53 FP
CORBIN, MURRAY D, 10550 QUIVIRA RD-5TH FL,66215
492-6200
39 M 1902 65 IM
COULTER, HENRY F, 4203 W 151 ST,66224
—
23 M 1902 51 OD
COULTER, THOMAS B, 8800 W 75TH SUITE 310,66204
677-3113
38 M 1205 64 OPH
COX JR, IRA, 5829 WOODSON RD BOX 975,66201
722-1100
19 M 1902 49 FP
CURRAN, KEVIN E, 4121 W 83RD,66208
649-9383
39 M 2803 65 OPH
DAVIS, RICHARD E, 8919 MISSION RD,66206
648-5303
26 M 1902 54 P
DELP, MAHLON H, 6131 TERRYDALE RD,66202
—
03 M 1902 34 IM
DENISON, TERRY R, 5811 OUTLOOK,66202
432-0625
29 M 1902 56 PD
DERRINGTON, KENNETH L, FOX HILL MED BLDG,66211
341-3535
44 M 1902 71 FP
DOCKHORN, ROBERT J, 5300 W 94TH TERR,66207
381-4674
34 M 1902 60 POA
DOHERTY, WILLIAM R, 7600 STATE LINE,66208
649-3900
20 M 3006 56 FP
DUCKETT II, THOMAS G, 4601 W 109TH SUITE 116,66211
648-1022
41 M 1902 67 OPH
EIKERMANN, WILLIAM C, 9400 MISSION RD,66206
642-5184
42 M 1902 69 P
ENDERS, WRAY, 9034 COTTONWOOD DR,66215
—
02 M 1902 36 ANES
ESRIG, HAROLD L, D.D., 8132 SAGAMORE,66206
381-5033
30 M 2878 60 ANES
ETZENHOUSER III, RUSSELL D, P O BOX 7426,66207
381-8282
34 M 1902 59 PD
EVANS JR, WILLIAM E, 8741 HIGH DRIVE,66206
362-7363
24 M 1902 58 FP
FERGUSON, ROBERT LEON, 8901 W 74TH SUITE 208,66204
362-6161
47 M 1902 73 IM
FORDYCE, NORMAN, 8901 W 74TH,66204
722-0020
41 M 1902 67 OTO
FRANCISCO, CLARENCE L, 3509 W 85TH,66206
371-6802
09 M 1902 34 DRS
FULLEN, JERYL G, 8901 W 74TH SUITE 124,66204
831-2604
43 M 401 68 ORS
GALLEHUGH, KEITH W, 9027 BIRCH,66207
371-4343
32 M 1902 57 R
GARCIA, FRANCISCO, 8020 SANTA FE,66204
642-5000
32 M 27501 60 FP
GARDNER, GLENN M, 5200 WEST 64TH,66208
—
35 M 2803 60 IM
GENTRY, KALE C, 5105 W 84TH,66207
632-4242
31 M 1902 60 FP
GILBERT, ROBERTA M, GEORGETOWN MEDICAL BLDG,66204
341-1234
35 F 3506 62 P
GILLEN, BILLY A, 8802 BIRCH LANE,66207
—
29 M 1902 54 ANES
GOLDSTEIN, GERALD L, 4601 W 109TH SUITE 120,66211
383-3630
47 M 15504 76 P
GOLLERKERI, MOHAN P, 7301 MISSION RD STE 339,66208
236-8866
30 M 49516 52 HEM
GOMEZ, FRANCISCO, 4200 SOMERSET #160,66208
649-7300
15 M 26401 43 P

GOOD, WENDELL LISLE, 4601 W 109TH,66211
649-3883
24 M 1902 48 FP
GOSALIA, ANIL V, 11701 MACKEY,66210
334-2500
46 M 49501 64 GP
GRAHAM, SUSAN B, 6423 RILEY,66202
-
F 1902
GROSSMAN, HARVEY M, 4601 W 109TH SUITE 122,66211
642-4040
49 M 1902 74 PD
GRUNOMEIER, ANNETTE M, 10550 QUIVIRA SUITE 250,66215
492-2013
46 F 1611 77 PD
HACKER, DAVID CHARLES, 6900 W 67TH,66202
588-6670
50 M 1902 75 ANES
HAMTIL, LAWRENCE W, 10550 QUIVIRA RD,66215
649-6722
36 M 2803 61 PD
HANDLEY, DENNIS MICHAEL, 8650 W COLLEGE BLVD,66210
649-1311
50 M 2803 76 FP
HARMS, ALBERT C, 5750 WEST 95TH,66207
381-5550
13 M 1902 38 FP
HARTONG, WILLIAM A, 8901 W 74TH STE 372,66204
831-9300
44 M 1902 71 IM
HASTINGS, MARY T, 2520 W 50TH PL,66205
-
45 F 1902 77 O
HATHAWAY, PETER, 11055 CEDAR STE 216,66211
383-2270
31 M 3503 60 IM
HEIDGEN, DEBRA L, 7906 KESSLER,66204
381-7845
55 F 1902 79 PD
HENRY, JOSEPH E, 8901 W 74TH STE 348,66204
432-8000
42 M 1902 68 PUD
HESSER, HERBERT H, 7207 EDGEWOOD BLVD,66203
-
06 M 1902 34 OD
HIEBERT, PETER E, 9021 SALEM DR,66215
-
03 M 1902 34 R
HILL, ROONEY W, 8901 W 74TH SUITE 208,66204
362-6161
47 M 1902 74 IM
HOBSON, MILBURN W, 9119 W 74TH ST,66204
831-2334
30 M 1902 55 OBG
HODES, HERBERT C, 5007 W 112TH,66211
381-6868
43 M 1902 69 OBG
HODGES, BRUCE E, DOCTORS BLDG SUITE 430,66215
888-0777
32 M 1902 63 FP
HOPKINS, WILLIAM O, 8800 W 75TH SUITE 350,66204
831-3500
33 M 2803 61 ORS
HORSEMAN, ROBERT F, 9119 W 74TH ST,66204
432-7419
19 M 1902 44 OBG
HOUSTON II, LAWRENCE MORLEY, 8650 W COLLEGE BLVD,66210
649-1311
50 M 2803 76 FP
HUANG, GEORGIANA L W, 6300 GLENWOOD #3,66202
384-4998
41 F 4107 75 GP
HUMPHREY, LOREN JENKINS, HUMPHREY MED CL & TUM INS,66204
384-2850
31 M 1611 56 SON
ITURRALDE, GEORGE, 7501 MISSION RD,66208
648-4949
21 M 13201 49 P
JACKSON, ROBERT V, 8901 W 74TH SUITE 150,66204
362-1660
49 M 2803 77 PD
JANES, DONALD R, 10550 QUIVIRA #360,66215
492-1955
34 M 1902 60 OBG
JOHNSON, NADINE KAY, 10550 QUIVIRA SUITE 510,66215
492-6200
38 F 1803 63 IM
JONES, CHARLES E, SHAWNEE MISSION MED CNTR,66201
676-2214
31 M 1902 60 FP
JONES, H IVOR, 8901 W 74TH SUITE 269,66204
362-4040
24 M 80303 51 P

JOUVENAT, NEIL C, 10550 QUIVIRA SUITE 120,66215
492-1844
43 M 3005 71 OBG
KADIAN, RAJESH S, 10550 QUIVIRA SUITE 260,66215
541-0577
50 M 71 IM
KAGAN, STUART M, 10550 QUIVIRA - SUITE 340,66215
492-1111
44 M 4901 69 PD
KARLIN, CHARLES A, PO BOX 4250,66204
677-0883
49 M 1902 75 OR
KARLIN, RONALD J, 9336 W 49TH, 66203
556-3000
54 M 1902 79 EM
KASHYAP, BANSHI PRASAD, 8901 W 74TH SUITE 257,66204
236-4500
47 M 49554 69 IM
KENNEDY, KENNETH R, 6100 MARTWAY STE 11,66202
432-0126
24 M 1902 53 FP
KETCHUM, LYNN O, 10550 QUIVIRA RD STE 310,66215
492-3737
36 M 2101 60 PS
KHURANA, SATISH K, 9119 W 74TH SUITE G-2,66204
432-3334
41 M 49536 65 PD
KIMURA, CHARLES C, 8901 W 74TH SUITE 125,66204
252-4220
25 M 2101 56 A
KODANAZ, A AYTEKIN, 5710 REINHAROT DR,66205
334-2500
28 M 90201 55 ANES
KOZIKOWSKI, BEN M, 7301 MISSION RD,66208
362-8317
30 M 2834 55 ORS
KRUEGER, KURT ALLEN, 10002 HOWE DR,66206
676-2479
48 M 3006 74 ANES
KUBIN, DORIS A, 2504 W 71ST,66208
-
15 F 1902 43 OD
KURTH, PAUL H, 5555 W 58TH,66202
432-2080
53 M 2507 77 IM
KURTH, ROBERT H, 5555 W 58TH,66202
432-2080
28 M 3005 53 IM
LAPI, RUTH M, 2012 STRATFORD RD,66208
-
14 F 4107 37 OD
LEATHERS III, HOLLIS K, SHAWNEE MISSION MED CNTR,66201
676-2340
38 M 3901 64 PATH
LEGASPI JR, PEDRO L, SHAWNEE MISSION MED CNTR,66201
676-2479
36 M 74801 60 ANES
LEIGH, LAWRENCE E, 8020 SANTA FE DR,66204
642-4080
12 M 1902 41 FP
LERNER, SCOTT A, 8901 W 74 SUITE 348,66204
432-8000
47 M 1902 73 PM
LESTER, JOHN BUCKLES, 4140 W 71ST SUITE 108,66208
432-7276
43 M 1902 70 P
LEWIN, WALTER, 8901 W 74TH ST,66204
362-4040
30 M 1902 56 P
LEWIS, JAMES E, 3700 W 83RD SUITE 203,66208
649-0923
37 M 2101 63 P
LIPSEY, JAMES H, 8800 W 75TH SUITE 350,66204
831-3500
31 M 1606 56 ORS
LOREN, MICHAEL LEWIS, 5300 W 94TH TERR,66207
381-4674
48 M 2507 73 PD
LULO, ANTONIO R, 7600 STATE LN,66208
649-3900
35 M 30801 60 IM
MALLORY, JOHN A, 10550 QUIVIRA SUITE 510,66215
492-6200
43 M 2803 71 IM
MANLEY, JOSEPH W, 4601 W 109TH SUITE 306,66211
381-8838
42 M 1902 69 OBG
MANTZ, FRANK A, 9309 W 103RD,66212
-
12 M 4101 38 OD
MARR, GARRETT L, 7312 ANTIOCH,66204
384-2850
44 M 1611 76 GS

MARVIN, NORMAN G, WYCLIFF SHOPPING CENTER,66212
541-8282
29 M 1902 56 FP
MASER, GEORGE R, 5808 NALL,66202
432-5515
12 M 1902 36 FP
MATHEWS, DAVID R, 3800 W 75TH ST,66212
432-9700
53 M 1902 78 FP
MATHEWS, ROBERT MAJOR, 7301 MISSION RD,66208
362-6888
25 M 1902 54 GS
MAXWELL, ROBERT A, 8901 W 74TH - SUITE 150,66204
362-1660
46 M 1902 73 PD
MCCAUGHEY, HUGH W, 11055 CEDAR SUITE 210,66211
381-1724
28 M 1902 53 IM
MCEACHEN, WILLIAM H, 3700 WEST 83RD SUITE 102,66208
649-3335
32 M 1902 59 PD
MCELROY, MICHAEL B, 6300 GLENWOOD,66202
432-1122
43 M 2803 69 ORS
MCGURK, THOMAS E, 4601 W 109 SUITE 206,66211
649-2080
39 M 2803 65 P
MCWHERTER, LOTTIE B, 5920 NALL SUITE 308,66202
362-1464
30 F 1902 57 IE
MENEZ, CESAR V, 4121 W 83RD SUITE 120,66208
381-4484
36 M 74810 56 P
MILLER, FREEMAN LANCE, 10550 QUIVIRA SUITE 340,66215
492-1111
48 M 1902 74 PD
MISKEW, DON B W, 7301 MISSION RD SUITE 348,66208
362-8317
42 M 5506 69 ORS
MOFFAT, ROBERT E, PO BOX 4250,66204
677-0883
42 M 1902 68 DR
MORONEY, JEAN M, 10550 QUIVIRA SUITE 510,66215
492-6200
25 F 4109 65 N
MUEHLBERGER, JAMES J, 4601 W 109TH SUITE 314,66211
383-3222
34 M 3006 60 PD
MUELLER, J KENT, 3700 WEST 83RD SUITE 203,66208
649-0923
35 M 1902 62 P
MURPHY, JAY W, 8901 W 74TH SUITE 208,66204
362-6161
49 M 3840 73 CD
MURRAY, W LEE, 4601 W 109TH SUITE 225,66211
381-2931
35 M 1902 61 OPH
NAUER, PAULA LOU, 7301 MISSION RD STE 342,66208
384-0745
49 F 1902 74 FP
NAVICKAS, LEONARD A, 8901 W 74TH SUITE 225,66204
362-5510
53 M 1902 77 FP
NEIBURGER, JAMES B, 4707 COLLEGE SUITE 100,62111
341-8907
45 M 1642 72 A
NELSON, BRYAN C, 9119 W 74TH,66204
384-5500
50 M 1902 75 PD
NELSON, JOHN B, 10550 QUIVIRA SUITE 510,66215
492-6200
48 M 2946 75 PUD
NIEMAN, JOHN L, PO BOX 7426,66207
381-8282
28 M 3806 58 PD
NOSTI, JUAN C, 8901 W 74TH #345,66204
262-5014
38 M 13204 63 PS
NYE, C ERIK, 7301 MISSION RD SUITE 348,66208
362-8317
39 M 3520 65 ORS
O'BRYAN, JAMES J, 10550 QUIVIRA,66215
492-2525
47 M 1902 73 PD
O'CONNELL, FRANK A, 7830 STATE LINE,66208
381-0685
25 M 1902 51 FP
OKTAWIEC, DANUTA, 5848 FONTANA DRIVE,66205
-
22 F 80303 50 ANES
OSGOOD, GEORGE M, 3700 WEST 83RD,66208
381-5200
14 M 1902 44 IM
PATTERSON, JOHN R, 5317 CHADWICK RD,66205
-
20 M 1902 48 PD
PEARCE, EUGENE W J, 9119 W 74TH SUITE 104,66204
722-3102
24 M 2802 49 OBG
PEARCE, LUNETTA M, 9119 W 74TH SUITE 104,66204
362-1525
26 F 3005 49 FP
PENTECOST, RICHARD L, 8900 STATE LINE SUITE 350,66206
383-1410
32 M 1001 56 P
PERICO, CARLOS J, 9209 ROE,66207
-
32 M 26404 60 FP
PETELIN, JOSEPH B, 9801 W 74TH SUITE 356,66204
677-2508
49 M 1902 76 GPVS
PETERSEN, A GENE, 3700 W 83RD,66208
648-3911
27 M 1902 54 IM
PETERSEN, GERALD D, 3700 WEST 83RD,66208
648-3911
30 M 1902 60 IM
PETIT, CARL ALFONSO, 10550 QUIVIRA SUITE 240,66215
888-1114
33 M 60 GS
PETRIE, SAMUEL C, GEORGETOWN MED BLDG,66202
722-1166
27 M 1902 58 IM
PFUETZE, BRUCE L, 4601 W 109TH,66211
383-3630
42 M 1902 68 A
PFUETZE, KARL D, 10550 QUIVIRA SUITE 510,66215
492-6200
40 M 1902 66 CD
PHILLIPS, WARREN G, 3700 W 83RD,66208
649-0923
26 M 1902 60 P
PILCHARD, WILLIAM A, GEORGETOWN MED BLDG,66204
362-3210
39 M 1602 65 OPH
PINGLETON, WILLIAM WARREN, 8901 W 74TH SUITE 348,66204
432-8000
42 M 3901 67 PUD
PITTS, RONALD L, 8901 W 74TH - SUITE 330,66204
362-2524
35 M 2002 62 D
POWELL, CAROL W, 8216 CHEROKEE CIRCLE,66206
381-3785
25 F 1902 51 P
POWELL, KENNETH A, 8216 CHEROKEE CIRCLE,66206
753-7000
25 M 1902 53 IM
PRENDES, CARLOS A, 6200 MASTIN,66203
677-5152
50 M 3005 79 GP
PRONKO, MICHAEL J, 4121 W 83RD SUITE 223,66208
648-7878
34 M 1902 60 P
RACELA JR, ANTONIO S, SUBURBAN MEDICAL CENTER,66215
492-1000
37 M 74802 63 PATH
RALSTIN, JAMES H, 14708 W 71ST TERR,66216
299-2069
49 M 1902 74 IM
REIVICH, RONALD S, 11030 GRANADA LANE,66211
383-3050
34 M 3806 60 P
RICE, BERNARD F, 8901 W 74TH SUITE 125,66204
262-4220
31 M 4113 56 END
RICHTER, DON G, SHAWNEE MISSION MED CTR,66201
676-2464
50 M 1902 79 ANES
RICK JR, GREGORY G, THE GEORGETOWN MED BLDG,66204
831-9300
40 M 1902 66 GE
RIEKE, FRANK A, 10411 WEST 55TH,66203
-
16 M 1902 41 OD
RIFFEL, LAWRENCE D, 10550 QUIVIRA SUITE 510,66215
492-6200
53 M 1902 78 IM
RITCHIE, KAREN S, 8901 W 74TH,66204
362-6161
49 F 3840 73 P
ROBERTSON, EDWARD J, SHAWNEE MISSION MED CTR,66201
676-2479
46 M 1902 78 ANES
ROBISON, JAMES T, 9800 W 75TH SUITE 310,66204
677-3113
21 M 4812 45 OPH

ROMITO, CYNTHIA L, 5811 OUTLOOK,66202
432-0625
47 F 1902 72 A
ROSENBERG, STANTON L, 1900 W 75TH SUITE 200,66208
362-8080
30 M 1902 55 P
ROSENTHAL, RICHARD, 5102 W 112TH ST,66211
492-1000
50 M 2820 76 EM
RUBIN, HERBERT M, 10550 QUIVIRA - SUITE 340,66215
492-1111
37 M 2802 63 PD
RUMOLO, MERVIN J, 6340 INDIAN LANE,66208
-
03 M 1902 30 GS
RYAN, MICHAEL E, 4601 W 109TH SUITE 210,66211
341-4030
46 M 1902 72 N
RYMER, ROBERT A, 8901 W 74TH SUITE 373,66208
722-0170
41 M 0702 68 OPH
SACHEN, FREDERICK L, 9620 ROE,66207
-
45 M 4813 79 N
SAFFO, KARL S, 8901 W 74TH,66204
362-9585
39 M 52801 62 PS
SATHYANARAYANA, SARASWATHI, 9119 W 74TH SUITE 206,66204
432-7419
45 F 49509 67 OBG
SAWKAR, LAXMIDAS A, 8901 W 74TH SUITE 312,66204
384-4844
36 M 49523 63 IM
SCHAEFER, JOSEPH PETER, 10550 QUIVIRA - SUITE 230,66215
492-7440
34 M 1902 60 IM
SCHROLL, JOHN T, 8901 W 74TH SUITE 248,66204
384-4990
51 M 1902 76 OBG
SCLAR, WILLIAM C, DOCTORS BLDG SUITE 450,66215
492-7730
46 M 2501 72 GS
SHAAD, DOROTHY J, 2322 W 51ST,66205
-
09 F 1902 44 OO
SHOFSTALL, WILLIAM H, 6100 MARTWAY,66202
722-4747
11 M 3901 41 ENT
SILLS, THOMAS D, 14809 W 85TH TERR,66215
281-8883
49 M 5606 77 EM
SILVER, BRADD J, 8901 W 74TH,66204
588-5048
50 M 1205 76 IM
SMITH, DALE C, 4501 W 109TH ST SUITE 224,66211
381-0353
20 M 1902 45 OPH
SMITH, DONALD J, 8600 W 95TH ST,66212
642-4515
18 M 1902 49 FP
SNODELL, FIRMIN E, 5555 W 58TH,66202
432-2080
31 M 1902 61 IM
SNOW JR, ARTHUR D, 8901 W 74TH - SUITE 225,66204
362-5510
45 M 1902 75 FP
SOELDNER, JAMES OLIVER, 8600 W 95TH,66212
341-0665
44 M 1902 70 FP
SPEER, FREDERIC, 5811 OUTLOOK,66202
432-0625
09 M 1902 34 A
STEINER, ROBERT M, 5311 W 96TH ST TERR,66207
677-0883
39 M 4112 65 R
STEINZEIG, ALFRED S, 6500 HODGES DR,66208
-
13 M 1902 37 OO
STEVENSON, E KENT, 4121 W 83RD SUITE 150,66208
649-5565
45 M 2802 67 CHP
STITT, RONALD W, 4221 W 104TH TERR,66207
342-7233
21 M 1902 45 J
STRIEBINGER, CHARLES M, 4601 W 109TH SUITE 307,66211
341-7299
45 M 1803 67 NS
STUBER, JACK LAWRENCE, PO BOX 4250,66204
677-0883
40 M 1902 66 OR
STUCKEY, CHARLES E, 10550 QUIVIRA SUITE 240,66214
492-7737
41 M 3005 68 GS

SUGAR, ROBERT L, 8901 W 74TH SUITE 248,66204
384-4990
40 M 3508 66 OBG
SULLIVAN, TOM G, 10550 QUIVIRA SUITE 320,66215
492-3600
44 M 1902 71 OBG
SULLIVAN JR, HENRY B, 5817 NIEMAN RD,66203
631-6160
24 M 1902 52 FP
SUTTON JR, RICHARD L, 3203 W 83RD TERR,66206
-
08 M 2501 29 OO
SZYMANSKI, DENNIS C, 10300 CATALINA,66207
561-4655
43 M 2507 70
TALLEY, ROBERT L, 6501 W 101 ST PLACE,66212
588-3000
48 M 4802 74 IM
TENNY, ROBERT T, 4601 W 109TH SUITE 307,66211
341-7299
51 M 1902 76 NS
TETZLAFF, ARCHIBALD D A, 4520 W 65TH,66208
334-2500
26 M 40721 52 ANES
THOMSEN, GARY, 8901 W 74TH SUITE 225,66204
362-5510
51 M 3005 76 FP
TOALSON, WILLIAM B, 8901 W 74TH SUITE 208,66204
371-3515
37 M 1902 63 CD
TRETBAR, LAWRENCE L, 8901 W 74TH,66204
677-1776
33 M 1902 60 GS
TROPP DO, ARNOLD L, 8600 W 95TH STE 102,66212
648-5600
45 M 2878 72 FP
TUCKER, SHERIDAN G, 4121 W 83RD SUITE 150,66208
649-5565
50 M 1902 75 P
TUTERA, GINO, 10550 QUIVIRA SUITE 120,66215
492-1844
45 M 2803 71 OBG
VALK, WILLIAM L, 5401 W 81ST,66208
588-6145
09 M 2501 37 U
VANNAMAN, DONALD D, P O BOX 7426,66207
381-8282
43 M 1902 71 PD
WALKER, WILLIAM L, 10500 QUIVIRA,66215
492-1000
44 M 1902 70 R
WANG, SIDNEY W, 10550 QUIVIRA SUITE 130,66215
492-1500
32 M 38503 58 FP
WEBB, JAMES R, 5349 NIEMAN ROAD,66203
631-0900
34 M 1902 61 FP
WERTH, CLAUDE J, 4121 W 83RD,66208
649-5566
37 M 1902 64 P
WHITEHEAD, RICHARD E, 7301 MISSION RD,66208
362-8317
31 M 2501 58 ORS
WHITLEY, DOUGLAS M, 4601 W 109TH SUITE 202,66211
341-4770
34 M 1902 60 D
WIGGINTON, GERALD D, D.O., 9119 W 74TH,66204
384-5500
44 M 2878 70 PD
WILEY, JOHN H, 9119 W 74TH SUITE 201,66204
831-2334
37 M 4113 63 OBG
WILSON, ROBERT B, 7301 MISSION SUITE 240,66208
362-3356
10 M 1902 40 ENT
WILSON, SLOAN J, 5618 W 62ND,66202
-
10 M 1902 36 HEM
WURSTER, GEORGE R, 3700 W 83RD SUITE 203,66208
649-0923
35 M 1902 61 P
YE, RICHARD C, 7301 MISSION RD,66208
362-7505
20 M 24222 46 PS
YOHE, RUTH M, 8600 W 95TH ST,66212
383-3377
25 F 4107 54 PDA
YOUNG, JOHN W, 8220 TRAVIS STE 115,66204
383-1550
37 M 4706 63 PS
YOUNGLOVE, R HAL, 10550 QUIVIRA SUITE 510,66215
492-6200
50 M 3005 75 OBG

YOUNGSTROM, KARL A. 9660 REEDER PLACE.66214
-
08 M 3607 44 OO
ZACK, ASHLEY S. 4601 WEST 109TH SUITE 122.66211
642-4040
45 M 2803 73 PO
ZAMIEROWSKI, DAVID S. 8800 W 75TH SUITE 340.66204
831-4113
42 M 2307 68 PS
ZIMMERMAN, DANIEL D. 4761 RAINBOW.66205
831-0910
45 M 3005 70 1M

SMITH CENTER—913
(Central Kansas Society)

RELIHAN, FRANCIS H. 116 SOUTH MAIN.66967
282-3291
84 M 1606 07 FP
SHEPPARD, ROBERT G. 120 E COURT.66967
282-6554
21 M 1902 45 GS
STEINKRUGER, VERLYN WILLIAM. 120 E COURT.66967
282-6554
28 M 3005 53 FP
WOODS, HUGH J. 120 EAST COURT.66967
282-6654
26 M 1902 52 FP

SOUTH HAVEN—316
(Tri-County Society)

UBELAKER, ERNEST J. .67140
892-2261
11 M 1902 38 FP

SPEARVILLE—316
(Barton County Society)

QUACKENBUSH, ROBERT P. SPEARVILLE DIST HOSP.67876
549-3471
21 M 1611 52 FP

ST. FRANCIS—913
(Northwest Kansas Society)

CRAM, ERNEST R. PO BOX 625.67756
332-2125
24 M 1902 52 FP
JEWELL, ROSS L. 203 SPENCER.67756
332-2832
13 M 1902 56 FP
STEPHENSON, LUCILLE C. .67756
-
06 F 1902 32 FP
WALZ, THOMAS J. 115 SOUTH QUINCY.67756
-
94 M 1902 21 OO

ST. JOHN—316
(Butler-Greenwood County Society)

SCHERER, ALFRED L. 610 E FIRST.67576
549-3261
32 M 1902 57 FP

ST. MARYS—913
(Pottawatomie County Society)

BROWN, FRED E. 602 W PALMER.66536
437-2255
25 M 1902 55 FP

STAFFORD—316
(Butler-Greenwood County Society)

BROWN, C EVERETT. 102 N MAIN.67578
234-5251
10 M 1902 47 A
WARD, ROBERT L. 412 E GRAND.67578
234-5281
24 M 1902 52 FP

STERLING—316
(Rice County Society)

OYSART, JACK C. 224 N FOURTH.67579
278-2181
12 M 3901 39 OO
SIMPSON, TOM C. 239 N BROADWAY.67579
278-2123
47 M 1902 73 FP

STOCKTON—913
(Central Kansas Society)

MAUCK, HAROLD C. 623 SOUTH 2ND.67669
425-6280
20 M 1902 54 FP
VOTAPKA, WILLIAM L. 623 S SECOND.67669
425-6280
24 M 1902 53 FP

SUBLETTE—316
(Southwest Kansas Society)

THIEMANN, A H. O.D., 301 DERBY.67877
675-2241
12 M 1805 42 FP

SYRACUSE—316
(Southwest Kansas Society)

FELDMEYER, SEELEY T. PO BOX 1087.67878
384-7735
46 M 74811 80 GP
PETTERSON, CECIL E. PROFFESIONAL ASSOCIATION.67878
384-5731
14 M 1902 39 FP

TONGANOXIE—913
(Leavenworth County Society)

STEVENS, PHILIP L. BOX 319.66086
845-2090
27 M 1902 54 FP

TOPEKA—913
(Shawnee County Society)

AGAN, LAWRENCE M. 1314 PEMBROKE LN.66604
357-4306
20 M 5002 44 R
ARREDONDO, MARIO. 6024 SW 26TH.66614
296-7215
25 M 26401 54 P
ARTZER, DENNIS C. 901 GARFIELD.66606
354-9591
51 M 1902 76 1M
ARUNAKUL, PUNYA. 1710 W TENTH.66604
234-2624
44 M 89104 69 OTO
ASHLEY, BYRON J. 3222 PLASS.66611
233-2280
98 M 1902 24 OPH
ASHLEY JR, B JOHN. 1616 WEST 8TH ST.66606
233-2280
31 M 1902 56 OPH
ATLURU, NARAYANA RAO. 631 HORNE SUITE 120.66606
232-8761
47 M 49550 69 ANES
AVERILL, STUART C. MENNINGER FD.66601
234-9566
24 M 502 52 P
BAEHR, RALPH H. S-E CAP REG RADIOTHER CTR.66606
234-3451
35 M 1606 59 R
BAIR, GLENN O. 534 MULVANE SUITE 400.66606
233-5153
31 M 2401 57 1M
BAKER, FREDERICK C. 2101 WEST 10TH.66604
232-0909
35 M 4113 62 FP
BAKER, PHILLIP L. 300 CONT MED BLDG.66606
357-0301
37 M 3005 63 ORS

BAKER, RAY D, 1515 W EIGHTH,66601
233-8961
30 M 4812 55 GPM
BARABAN, MARC R, 1319 HUNTOON,66604
357-5325
50 M 2846 75 PS
BARNHILL, C ALTON, MENNINGER FD,66601
234-9566
31 M 3501 56 P
BAUCOM, KARAN YVONNE, 1516 W SIXTH,66604
233-1639
50 F 1902 75 DBG
BAUCOM-COPELAND, SHARON LAVARNE, 634 MULVANE STE 402,66606
235-9545
50 F 2846 75 FP
BAUDE, EUGENE L ANDRE, 1815 WESTWOOD CIRCLE,66604
354-1946
02 M 39606 27 IM
BEACH, RICHARD R, 106 MED ARTS BLDG WEST,66604
233-7943
23 M 2802 48 1M
BEALE, DAVID A, MENNINGER FD,66601
234-9566
31 M 5404 56 P
BEATY, JAMES R, 1700 WEST 7TH,66606
354-1111
32 M 5101 66 EM
BECK, JOSEPH D, 2760 SW BURLINGAME,66611
-
18 M 3005 43 OD
BEDFORD, D R, PO BOX 1772,66615
-
09 M 4802 40 1M
BEELMAN, FLOYD C, 1286 LAKESIDE DR,66604
-
02 M 3840 35 FP
BELLER, WILLIS L, 3651 HOLLY LANE,66604
-
14 M 1902 41 OD
BERLAND, DAVID I, BOX 829,66601
234-9566
47 M 2803 78 P
BLAKE, HENRY S, 1933 WESTWOOD DR,66604
-
11 M 3520 37 OD
BONEBRAKE, C RICHARD, 634 MULVANE STE 104,66606
233-1979
48 M 1606 75 DBG
BOREL, DAVID, ST FRANCIS HOSP & MED CTR,66606
295-8473
45 M 1902 71 PATH
BOWEN, CLOVIS W, 2200 WEST 10TH,66604
234-8601
12 M 1902 37 FP
BOWEN JR, HARRY J, 2200 WEST 10TH,66604
234-8601
11 M 1902 37 FP
BOYD, SPENCER H, 1815 WEST 2ND,66606
-
11 M 1902 35 DBG
BRAHMAN, HERBERT D, 1700 SEVENTH,66606
295-8471
43 M 512 70 PATH
BRAUN, ROBERT W, 901 GARFIELD,66606
354-9591
44 M 2803 70 1M
BRAUNSDORF, ROBERT L, 2625 DHID,66605
354-7630
08 M 2501 35 FP
BRIDWELL, RUSSELL E, 1710 W TENTH,66604
234-2524
26 M 1902 51 ENT
BURDZIK, EBERHARD G, 2700 W SIXTH,66606
296-4222
25 M 40916 54 P
BYLANDER DO, TERESA I, 3556 SW CAMBRIDGE,66614
272-3111
46 F 1875 77
CACHIA, RICHARD M, DEPT OF PATHOLOGY,66606
295-8472
51 M 62701 73 PATH
CARVER, LARRY A, 130 CONT MED BLDG,66606
234-8653
37 M 1902 73 P
CASHMAN JR, MAURICE R, 901 GARFIELD,66606
354-9591
35 M 1902 61 HEM
CASTRO, JUAN A, 3107 W 21ST,66604
295-5401
34 M 4201 64 PD
CAVANAUGH, JOHN W, 200 PROFESSIONAL BLDG,66604
235-3488
13 M 1803 39 GS

CHAMBERLIN JR, CECIL R, BOX 829,66601
234-9566
30 M 3901 55 CHP
CHANG, FONG CHUNG, 303 MEDICAL ARTS BLDG,66604
235-3451
40 M 38505 68 ANES
CHEN, CHU-CHI, 918 W TENTH,66604
354-4465
47 M 24405 73 U
CHEN, TAK-MING, 303 MED ARTS BLDG,66604
234-3451
41 M 24402 68 ANES
CHERRY JR, ARTHUR C, 918 WEST 10TH,66604
233-3362
27 M 3806 53 PD
CLARK, CRAIG N, 3124 E 6TH,66607
354-7683
29 M 1902 58 FP
COCHRAN, PAUL W, MENNINGER FD,66601
234-9565
33 M 4802 58 1M
COHEN, LOUIS, 918 W 10TH,66604
233-7175
14 M 1902 41 1M
COKELEY, JOHN M, 2200 GAGE BOULEVARD,66622
272-3111
30 M 5101 55 P
COKER, W LAURENCE, 631 HORNE #340,66606
232-9354
53 M 1902 78 FP
COLLINS, DEAN T, MENNINGER FD,66601
234-9566
28 M 1902 55 P
COLLINS, EDWARD JOSEPH, 900 WASHBURN,66606
233-3242
45 M 1611 71 OPH
COLLINS, ELISABETH B, 2400 W 29TH,66611
257-0150
21 F 40905 46 P
COLLINS, FRANCIS T, 206 MED ARTS BLDG EAST,66604
233-6470
14 M 1902 43 1M
CONNELLY, JOHN C, MENNINGER FD,66601
234-9566
39 M 3806 65 P
CONROY, ROBERT W, MENNINGER FD,66601
234-9566
38 M 2504 64 P
COOLEY, DENNIS M, TOPEKA MEDICAL CENTER,66604
233-3362
51 M 1902 77 PD
COTTON, ROBERT T, 901 GARFIELD,66606
354-9591
19 M 1902 45 1M
CRARY, JOHN E, 201 MED PLAZA BLDG,66604
233-4202
18 M 1902 43 1M
CROUCH, STEVEN W, 904 MULVANE, 66606
232-8224
51 M 1902 76 PD
CROUCH, WILLIAM H, 904 MULVANE,66606
232-8224
20 M 2802 45 PD
DAVIS, CHESTER R, 631 HORNE,66606
232-9394
50 M 1902 75 FP
DELCORE, ROMANO, BOX 829,66601
234-9566
30 M 54901 50 P
DELGADO, SERGIO, 634 MULVANE STE 200,66606
357-0352
37 M 2501 62 ORS
DESOIGNIE, RAFAEL R, 3107 W 21ST,66604
296-5305
19 M 27501 P
DONEPUDI, RAO S, 631 HORNE #120,66606
232-8761
49 M 49550 74 ANES
DUNAGIN, JACK A, TOPEKA PSYCHIATRIC CTR,66601
233-7138
20 M 1902 44 P
DUNIVEN, PHILIP L, RADIOLOGY & NUCLEAR MED,66604
234-3451
52 M 4812 77 R
DURST JR, ROBERT D, CONTINENTAL MED BLDG,66606
357-5166
42 M 2803 69 D
EATON, EDWARD L, TOPEKA PSYCHIATRIC CTR,66601
233-7138
40 M 0401 73 P
ELDER, DOUGLAS M, 310 MED ARTS BLDG,66604
234-3451
41 M 1902 69 DR

FAIRCHILD, RICHARD S, 901 GARFIELD,66606
354-9591
48 M 1902 76 IM
FEAGAN, JERRY, 901 GARFIELD,66606
354-9591
39 M 1902 63 GE
FERNANDEZ, LUIS A, PO BOX 118,66601
233-8961
14 M 27501 41 PD
FIELD, RICHARD A, 303 MEDICAL ARTS BLDG,66604
235-3451
29 M 1902 55 ANES
FILLMAN, ELDON M, 301 MEDICAL PLAZA BLDG,66604
233-4256
20 M 1611 44 U
FORD, FRED L, 220 MEDICAL ARTS BLDG,66604
234-5516
11 M 2501 36 GS
FOSTER, CHARLES G, 110 CONT MED BLDG,66606
232-6964
21 M 4102 47 IM
FOSTER, D BERNARD, 3111 JEWELL,66611
-
14 M 2501 38 NP
FUSILLO, MICHAEL, BOX 829,66601
234-9566
48 M 3508 74 P
GANDHI, SHANTIKUMAR K, 40 MED ARTS BLDG,66604
233-1710
40 M 49501 67 TS
GANZARAIN, RAMON C, MENNINGER FD,66601
234-9565
23 M 23101 47 P
GAY, JOHN D, 310 MED ARTS BLDG,66604
234-3451
42 M 4802 68 DR
GENDEL, JOSEPH E, 918 W 10TH,66604
235-9914
12 M 4804 37 ORS
GIESSEL, MICHAEL D, CONTINENTAL MED BLDG,66606
357-5166
48 M 1902 74 D
GIMPLE, KENNETH, 631 HORNE #200,66606
233-7491
45 M 1902 71 ORS
GLEASON, JIMMIE A, 800 LINCOLN,66606
233-5101
33 M 1902 58 OBG
GODFREY, KENNETH E, 9550 SW 53RD,66604
273-5153
18 M 3901 50 P
GOOTEE, JOSEPH E, 3204 W 17TH,66604
-
21 M 2834 47 OO
GRAHAM JR, CHARLES P, 631 HORNE #400,66606
354-9504
40 M 3601 65 GS
GRAY, DAVID E, PO BOX C-50,66601
234-8621
16 M 1506 42 GEN
GRAYBB, ANTOINE S, MEMORIAL HOSP,66607
354-5100
18 M 60501 46 IM
GREENBERG, MARK, 310 MED ARTS BLDG,66604
234-3451
46 M 1611 72 R
GREENE, HORACE T, 1710 W 10TH,66604
354-7508
15 M 401 42 FP
GREENWOOD, EDWARD D, 3617 W 6TH,66601
-
01 M 702 38 CHP
GREER, RICHARD H, 918 W 10TH,66604
-
09 M 1902 39 OO
GUTOVITZ, ALLEN LOUIS, BROCK MEDICAL PLAZA,66606
233-9643
46 M 1611 72 CD
HACKER, ELAINE MARY, 3026 QUAIL CREEK,66614
296-3981
25 F 2504 50 OBG
HAINES, ROBERT A, 2700 SIXTH,66606
296-3436
16 M 3841 42 P
HALLEY, M MARTIN, 40 MED ARTS BLDG,66604
233-1710
27 M 2401 53 TS
HARRIS, HUBERT L, 210 MED ARTS BLDG,66604
233-3151
12 M 1803 39 D
HARRIS, PATRICIA A, 1617 W 26TH,66611
354-1905
29 F 1902 54 IM

HARRISON, HALL E, 901 GARFIELD,66606
354-9591
39 M 2802 65 IM
HEBBAR, SATYA N, BROCK MEDICAL PLAZA,66606
233-9543
39 M 49509 62 CD
HERRERA, JORGE J, 2825 CALIFORNIA,66605
354-5100
27 M 64901 55 IM
HIEBERT, JOHN B, 901 GARFIELD,66606
354-9591
40 M 1902 68 CD
HILL, ROBERT N, 901 GARFIELD,66606
354-9591
14 M 1902 67 IM
HILST, WILBUR D, 1271 WOODHULL,66614
273-5870
31 M 3005 55 GS
HIRSCHBERG, J COTTER, MENNINGER FD,66601
234-9566
15 M 1602 40 CHP
HISZCZYNSKYJ, ROMAN, 1500 W TENTH,66604
354-6870
35 M 1803 66 PATH
HOBBS, DONALD D, 200 CONTINENTAL MED BLDG,66606
233-7491
28 M 2401 54 ORS
HOHERZ, DAVID G, 631 HORNE SUITE 220,66606
235-1170
45 M 1902 76 TS
HOLMES, ROBERT W, 901 GARFIELD,66606
354-9591
52 M 1902 74 IM
HORNE, JAMES B, 1305 LAKESIDE DR,66604
233-1730
25 M 1506 52 P
HOSTETTER, JAMES P, 1500 W TENTH,66604
354-6100
43 M 1902 69 EM
HOYT, ARTHUR W, 2055 CLAY,66604
234-5663
14 M 2501 40 P
HSU, CHENG H, 1516 W SIXTH,66606
233-9681
41 M 38502 66 U
HSU, SHIN-FU, MEDICAL PLAZA BLDG,66604
232-0362
43 M 24402 68 OTO
HUANG, JONSON, 901 GARFIELD,66606
357-6171
52 M 2701 77 N
HUSTON, JOSEPH W, 634 MULVANE,66606
357-0352
35 M 1902 62 ORS
HUTTON, FREDERICK A, 102 MED PLAZA BLDG,66604
234-0553
29 M 6701 58 PS
HYLAND, JOSEPH M, BOX 829,66601
234-9565
45 M 53902 68 P
ILIFF, R DOUGLAS, 1500 SW OAKLEY,66604
354-6100
49 M 1902 74 FP
ILORETA, ALFREDO T, 6950 SW 33RD,66614
272-3111
47 M 74801 71 U
ISAACSON, RICHARD N, 1001 GARFIELD STE 301,66604
233-4255
48 M 2501 75 U
JACKSON, LINDA H, 212 WOODLAWN AVE,66606
234-9566
42 F 3601 67 CHP
JACOBY II, ROBERT E, 340 CONT MED BLDG,66606
232-9394
46 M 2307 72 FP
JANSSEN, ERWIN T, MENNINGER FD,66601
234-9565
35 M 1803 62 P
JOHNSON, EDEN E, 5020 SW 28TH,66614
234-5481
39 F 74808 61 P
JOSEPH, BRIAN W, 130 CONT MED BLDG,66606
234-8653
38 M 35205 61 CHP
JOSS, CHARLES S, 221 MED ARTS BLDG W,66604
232-0444
14 M 1606 40 GS
JOYCE, G BERNARD, 4929 WEST HILLS DR,66606
-
17 M 1902 44 ORS
KATZ, JEROME B, MENNINGER FD,66601
234-9566
22 M 2101 44 P

KAVEL, KARL K, MED PLAZA BLDG,66604
234-2663
36 M 3505 64 PDA
KELLY, DAN A, 904 MULVANE,66606
232-8224
39 M 2803 64 PD
KENNEDY, HOWARD U, 1115 W TENTH SUITE A,66604
233-8268
18 M 401 44 IM
KEYS JR, ROBERT C, 303 MED ARTS BLDG,66604
235-3451
35 M 1902 62 ANES
KEYS SR, ROBERT C, 3034 LYDIA,66614
97 M 1902 27 OD
KIM, YONG W, CONT MED BLDG,66606
232-6964
28 M 58302 49 IM
KINDLING, PAUL H, 40 MED ARTS BLDG,66604
233-1710
30 M 3545 61 TS
KIRKEGAARD, ROGER S, 835 WESTERN,66606
233-6493
30 M 1803 56 OPH
KLEINHOLZ JR, EMIL JOHN, 600 MADISON,66607
354-5284
39 M 3503 65 IM
KLEMMER, HERBERT, MENNINGER FD,66601
234-9566
11 M 4102 37 P
KOVARIK, ERNEST D, 900 WASHBURN,66606
357-5171
36 M 3005 64 DPH
KROLL, HARRY G, 200 CONTINENTAL MED BLDG,66606
233-7491
24 M 1602 50 ORS
LACCHEO, MICHAEL L, 2115 W 10TH,66604
51 M 3340 76 FP
LAI-CHEN, SHEUE-HUEY, 918 W TENTH,66604
354-4465
48 F 38505 73 GP
LAUNEY, WALTON S, 1001 HORNE #310,66604
234-3451
39 M 4804 75 R
LAWSON, DWIGHT, 108 MEADOW LANE,66606
06 M 2802 30 IM
LEE, CATHERINE, 600 MADISON,66607
354-5224
49 F 2307 75 IM
LEE, SONG OOW, 303 MED ARTS BLDG,66604
235-3451
43 M 38505 68 ANES
LEE, SONG PING, 918 W 10TH,66604
233-6001
34 M 38502 61 OTO
LEGLER DO, GARY LEE, 2835 SE SKYVIEW CT,66605
233-8961
50 M 2878 77 GP
LEIFER, WILLIAM N, 1500 W TENTH,66606
354-6031
47 M 1902 78 PATH
LENTZ, WILLIAM R, 308 MED ARTS BLDG,66604
235-3443
24 M 1902 53 FP
LESSENDEN JR, C M, 5635 NW BRICKYARD RD,66618
272-3111
18 M 1902 43 D
LEVY, EDWIN Z, 4125 SW GAGE CTR OR L-#6,66604
273-5610
29 M 1506 54 P
LIESMANN, GEORGE E, 631 HORNE,66606
235-1170
49 M 1902 74 GPVS
LIESMANN, JEAN ELIZABETH, MEMORIAL HOSP,66607
354-5275
49 F 1902 74 IM
LONG, JOHN W, 1001 HORNE ST,66604
233-1710
43 M 1902 70 TS
LUMB, RAYMOND C, 901 GARFIELD,66606
354-9591
42 M 1001 68 RHU
LYNCH, JOHN A, 300 CONT MED BLDG,66606
357-0301
30 M 2834 55 ORS
MARSHALL, B M, 1826 SW 34TH,66611
08 M 2802 34 U
MARTIN, WILLIAM O, 303 MED ARTS BLDG,66604
235-3451
19 M 1902 44 ANES

MAU, WALTER, 301 MEDICAL PLAZA BLDG,66604
233-4255
15 M 1611 40 U
MCCARTER, DUANE K, 2101 W 10TH,66604
233-8979
26 M 1902 58 IM
MCCLELLAN, JOHN W, 2701 BOSWELL,66611
11 M 3006 36 OO
MCCLURE, JAMES A, 1541 WESTOVER RD,66604
18 M 1902 44 U
MCCOMAS JR, MARMADUKE D, 3020 BRUSH CREEK CIRCLE,66614
272-2535
16 M 1902 43 U
MCCOY, MICHAEL T, 631 HORNE SUITE 300,66606
357-0301
49 M 1902 75 ORS
MCELROY, ROBERT T, 221 MED ARTS BLDG,66604
232-0444
35 M 1902 61 GS
MCELROY, WILBUR J, 1616 W EIGHTH,66606
233-2280
35 M 1902 61 OPH
MECH, ARNOLD W, 3240 TIMBERLAKE LN,66614
234-9565
52 M 1643 77 P
MEGIBOW, ALAN D, TOPEKA PSYCHIATRIC CTR,66601
233-7138
37 M 3503 64 CHP
MEIDINGER, RICHARD, 310 MED ARTS BLDG,66604
234-3451
39 M 1902 65 DR
MENNINGER, KARL A, BOX 829,66601
93 M 2401 17 P
MENNINGER, ROBERT G, 3617 W 6TH ST,66601
234-9565
22 M 3545 52 P
MENNINGER, ROY W, 3617 W 6TH,66601
234-9566
26 M 3520 51 P
MENNINGER, W WALTER, THE MENNINGER FD,66601
234-9566
31 M 3520 57 P
MEYER, O WARREN, 634 MULVANE #100,66606
233-9643
49 M 1902 74 CO
MILLS JR, PHILIP E, 901 GARFIELD,66606
357-6171
36 M 1902 64 N
MITTLEMAN, FREDERICK S, MENNINGER FD,66601
234-9566
45 M 3006 70 P
MOOLIN, HERBERT C, MENNINGER FD,66601
234-9565
13 M 3005 38 P
MOORE, HUGH C, 1500 W TENTH,66606
354-6031
33 M 4812 59 PATH
MORRIS, MERLE O, 1401 W 10TH,66604
234-2377
21 M 1902 45 OBG
MORRISON, MICHAEL R, 800 LINCOLN,66606
233-5101
50 M 1902 76 OBG
MORROW JR, J TARLTON, 235 WOODLAWN,66606
23 M 4804 47 CHP
MUELLER, ARNOLD V, 901 GARFIELD,66606
354-9591
31 M 3005 57 IM
MURPHY, THOMAS MEAD, PO BOX 829,66601
234-9566
41 M 3901 68 P
MYERS, JO ANN, MENNINGER FD,66601
234-9565
28 F 1902 53 P
NABOURS, RICHARD D, 4228 W 29TH ST TERR,66614
272-7190
27 M 1902 54 FP
NANCE, JOEL, MENNINGER FOUNDATION,66601
234-9566
42 M 3546 72 P
NICE, G WILLIAM, 112 MED ARTS BLDG EAST,66604
235-8090
22 M 1902 46 IM
NOVOTNY, PETER C, MENNINGER FD,66601
234-9565
30 M 15407 55 P
NYSTROM, CURTIS A, 2707 W 29TH,66614
272-8440
23 M 1902 54 FP

O'NEIL, ROBERT H, 901 GARFIELD,66606
3S4-9S91
20 M 1902 45 IM
OBOURN, ROBERT L, 1150 OAKLEY,66604
234-9565
19 M 2802 50 ?
PALMBERG, KENT E, 901 GARFIELD,66606
3S4-9591
49 M 1902 74 IM
PARMAN, ROBERT D, 904 MULVANE,66606
232-8224
27 M 1902 54 PD
PARULKAR, DEEPAK S, 631 HORNE SUITE 120,66606
232-8761
49 M 49S17 73 ANES
PATEL, VINOO, PO BOX 2401,66601
3S7-6171
47 M 49S31 69 N
PATINO, EDGAR, MENNINGER FD,66601
234-9S66
41 M 26406 66 ?
PATRICK, FRED EDWARD, 904 MULVANE,66606
232-8224
4S M 1902 71 PD
PAYNE, ROBERT R, 200 CONTINENTAL MED BLDG,66606
233-7491
29 M 1902 5S ORS
PENN, GEORGE M, VETERANS MEDICAL CENTER,66622
272-3111
30 M 4802 S8 ?
PERDUE II, W LANG, 631 HORNE #400,66606
3S4-9504
49 M 1902 74 GS
PETERSON, DEAN L, 303 MED ARTS BLDG WEST,66604
235-34S1
24 M 1902 S4 ANES
PETERSON, ROBERT L, STORMONT-VAIL EMERGENCY,66606
3S4-6108
35 M 1902 62 EM
PETERSON, VERNON J, 310 MED ARTS BLDG,66604
234-34S1
42 M S12 68 R
PETRIK, EDWIN L, MEMORIAL HOSPITAL,66607
3S4-S100
3S M 1902 64 IM
PETTERSON, DENNIS CRAIG, 310 MEDICAL ARTS BLDG,66604
234-34S1
49 M 1902 74 R
PFUETZE, ROBERT E, 209 MED ARTS BLDG EAST,66604
-
09 M 1902 35 OBG
PIERCE, CHARLES F, 918 W 10TH,66604
23S-6282
24 M 4101 S1 OBG
PIERCE, DONALD R, 307 MED ARTS BLDG EAST,66604
235-2226
23 M S101 49 FP
POLLY, RICHARD E, 631 HORNE ST,66606
3S7-0301
42 M 1803 68 ORS
PORTER, ROBERT D, 901 GARFIELD,66606
3S4-9S91
41 M 2802 67 IM
POWELL, WILLIAM R, 833 GARFIELD,66606
233-8941
30 M 1902 S4 GS
POWELL II, BENSON M, 400 CONTINENTAL MED BLDG,66606
354-9S04
26 M 1506 49 TS
PRESTON, RALPH R, 1710 WEST 10TH,66604
234-2624
19 M 1902 44 OPH
PROKOP, BRADFORD S, 900 WASHBURN,66604
233-3900
32 M 1606 S7 OPH
PYLE, LUCIEN R, 3139 CANTERBURY LN,66604
-
01 M 1601 28 OBG
RAINBOW-EARHART, KATHRYN A, 2916 KENTUCKY,6660S
233-1730
21 F 4707 48 P
RAJU, A S PADMA, 918 W 10TH,66604
234-3447
39 M 49S09 61 TS
RAMSEY, BARTLETT W, 904 MULVANE,66606
232-8224
2S M 1902 50 PD
RANSDELL, EDGAR C, 800 LINCOLN,66606
233-S101
41 M 3005 66 OBG
RANSOM, JAMES H, MEDICAL PLAZA BLDG,66604
234-2663
36 M 1803 62 A

REINKING, VICTOR E, 918 WEST 10TH,66604
233-5084
26 M 1902 S1 IM
REYMOND, RALPH O, S-F CAP REG RADIOTHER CTR,66606
234-34S1
37 M 2301 67 R
RICCI, ROBERT LAWLER, 901 GARFIELD,66606
354-9591
50 M 1902 7S IM
RICH, JOSEPH E, 634 MULVANE SUITE 104,66606
233-1979
47 M 40734 74 OBG
RICHARDSON, J M, 634 SW MULVANE,66606
233-9681
47 M 1611 74 IM
RIEDEL, ROBERT H, 6731 SW ALYESBURY RD,66610
235-0011
04 M 2802 28 PH
ROBERTS, WARREN E, PO BOX 4047,66604
272-S797
25 M 1902 S7 FP
ROBINSON, DAVID B, 800 LINCOLN,66606
233-S101
47 M 1902 73 OBG
ROEDER, ROBERT E, 901 GARFIELD,66606
3S4-9S91
40 M 1902 67 IM
ROSS, JACK L, MENNINGER FD,66601
234-9566
32 M 4812 56 P
ROTERT, LARRY, 301 MEDICAL PLAZA BLDG,66604
233-42S5
38 M 3007 66 U
ROY, WILLIAM R, 634 MULVANE STE 104,66606
233-1979
26 M 1606 48 ADM
RUCKER, CLEMENS, 120S W 29TH,66611
-
86 M 2822 05 OO
RUNNELS, JOHN B, 901 GARFIELD,66606
3S7-6171
35 M 1902 61 NS
RUPP, RICHARD J, 901 GARFIELD,66606
3S4-9591
42 M 3941 68 CO
SANCHEZ, ROGELIO, 1516 W 6TH ST,66606
232-100S
31 M 64901 64 U
SARGENT, JOSEPH O, MENNINGER FD,66601
234-9S65
32 M 2S01 S8 IM
SAYLOR, EDWARD H, 918 WEST 10TH,66604
233-3362
39 M 1902 6S ?O
SAYLOR, LESLIE L, 918 WEST 10TH,66604
-
07 M 1606 35 OO
SAYLOR, MARK, 918 WEST 10TH,66604
234-3211
37 M 1902 66 GS
SAYLOR, STEPHEN, 631 HORNE #340
232-9394
47 M 1902 73 FP
SCAMMAN, W WIKE, PO BOX C-S0,66601
234-8621
32 M 4705 S7 PATH
SCHLOESSER, HARVEY L, 918 MERCHANTS NATL BK,66612
23S-3184
21 M 3901 S1 P
SCHLOESSER, PATRICIA T, 1914 WARNER CT,66604
862-9360
24 F 3901 49 PO
SCHRAM, PETER CHARLES, 130 CONTNENTAL BLDG,66606
234-86S3
39 M 2S07 69 P
SEGERSON, JOHN A, 901 GARFIELD,66606
357-6171
1B M 3S4S 43 N
SEGURA, ANTONIO, 2321 W 33RD,66611
272-3111
30 M 26401 54 ANES
SEHOEV, JOAN, MEMORIAL HOSP,66607
354-S100
40 F 5101 63 FP
SETTLE SR, RUSSELL O, 2320 CHELSEA DRIVE,66614
-
04 M 1902 29 OO
SHAW, JOSEPH L, MED ARTS BLDG #204,66604
23S-6221
34 M 511 60 ORS
SHEAFOR, DOUGLAS, TOPEKA PSYCHIATRIC CTR,66601
233-7138
34 M 1902 60 P

SHELTON, STEPHEN E, TOPEKA PSYCHIATRIC CTR,66601
233-7138
35 M 702 61 P
SHERWOOD JR, CLARENCE E, CONTINENTAL MED BLDG,66606
354-9504
22 M 702 53 GS
SIMPSON, WILLIAM S, MENNINGER FOUNDATION,66601
234-9566
24 M 6001 48 P
SISK, PHILLIP B, 310 MED ARTS BLDG WEST,66604
234-3451
32 M 1803 56 R
SMITH, LEO A, 918 BUCHANAN,66604
-
08 M 3006 33 OD
SNARR, JACK W, MED ARTS BLDG W #310,66604
234-3451
41 M 6201 65 DR
SPEARMAN, JESSE L, MED ARTS BLDG ROOM 24,66604
234-2879
20 M 1902 54 OBG
SPENCER, MILLARD C, 310 MED ARTS BUILDING,66604
234-3451
28 M 1902 55 R
SPENCER, WAYNE E, 103 MED ARTS BLDG EAST,66604
233-9686
38 M 1902 64 IM
STEIN, JOSEPH M, 901 GARFIELD,66606
357-6171
24 M 3519 47 N
STEINER, FREDERICK T, 3251-H RANDOLPH,66611
234-9566
50 M 5606 76 IM
STOCK, KARL W, 1710 W 10TH,66604
235-5205
13 M 2834 37 OPH
SUFI, M ASHRAF, 1001 HORNE #111,66604
354-8518
43 M 70402 68 IM
SUTTON, RICHARD O, 1706 W TENTH,66604
235-2311
38 M 4706 67 ORS
SWOGGER JR, GLENN, MENNINGER FO,66601
234-9566
35 M 3806 60 P
TAPPEN, DANIEL L, 2849 MACVICAR,66611
-
16 M 1902 41 OD
TARGOWNIK, KARL K, 1218 W TENTH,66604
296-7281
15 M 40710 49 P
TARNOFF, GERALD M, BOX 829,66601
234-9566
48 M 1542 73 P
TARNOWER, WILLIAM, MENNINGER CLINIC,66601
234-9565
21 M 4802 48 P
TEMPERO, STEPHEN J, 310 MED ARTS BLDG,66604
234-3451
42 M 1606 67 R
THOMS, NORMAN W, 40 MED ARTS BLDG,66604
233-1710
34 M 2501 59 TS
THURSTON, DAVID E, 200 CONTINENTAL BLDG,66606
233-7491
29 M 1902 55 ORS
TIETZE, DENNIS D, 634 MULVANE STE 402,66606
235-9545
50 M 1902 78 FP
TOTH, JOHN ROY, 2115 W TENTH,66604
232-3330
49 M 1902 71 FP
TOZER, RICHARD C, 1447 OAKLEY,66604
-
19 M 4102 45 OD
TRAVIS, JOHN W, S-F CAP REG RADIOTHER CTR,66606
295-8000
29 M 1606 55 R
TREES, CLYDE B, 3700 HUNTOON,66604
-
09 M 2401 33 OD
TREGER, NEWMAN V, 1704 W 10TH,66604
354-8761
16 M 1902 40 IM
TUCKER, VIRGINIA L, BUREAU OF MAT & CHILD HLT,66620
862-9360
30 F 1902 57 PD
TWEMLOW, STUART W, 707 QUINCY SUITE 208,66603
233-1607
41 M 67101 73 P
UHR, NATHANIEL, MENNINGER FOUNDATION,66601
234-9566
00 M 3519 21 IM

VAN SICKLE, GREGGORY J, 634 MULVANE,66606
233-8508
49 M 1506 75 PD
VANOE GARDE, LARRY D, 800 LINCOLN,66606
233-5101
41 M 1803 66 OBG
VOGEL, STANLEY J, 901 GARFIELD,66606
354-9591
44 M 2802 70 IM
VOTH, HAROLD M, MENNINGER FOUNDATION,66601
234-9566
22 M 1902 47 P
WADE III OD, WILLIAM E, PO BOX 1531,66601
272-3111
53 M 3979 80 P
WALLACE, LED F, 5500 W 24TH,66614
-
17 M 1902 41 OD
WALLS, WILLIAM J, 310 MED ARTS BLDG,66604
354-6171
39 M 2834 66 DR
WALZ, ROYCE C, 1710 W 10TH SUITE 205,66604
234-2676
27 M 15407 60 P
WANLESS, KIRK M, 1424 W EIGHTH,66606
232-8188
44 M 2803 74 OTO
WARD, HOWARD N, 901 GARFIELD,66606
354-9591
37 M 1606 62 HEM
WARE, LUCILE M, MENNINGER FOUNDATION,66601
234-9566
29 F 3501 53 P
WARRICK, DAVID ALAN, 600 MADISON,66607
354-5275
49 M 3843 76 IM
WATERS, DALE A, 1001 HORNE #40,66604
233-1710
41 M 5605 67 CDS
WEAVER, WALTER D, 900 WASHBURN ST,66606
233-3636
41 M 1902 69 OPH
WEBER, DARRELL J, 1710 W 10TH,66604
233-2305
16 M 1902 44 FP
WELSH, NANCY JANE, 5437 SW 12TH ST TERR,66604
296-7281
39 F 3840 63 EM
WIKSTEN, VERNON C, 807 TERRACE AVE,66611
-
11 M 1902 37 OD
WILCOX, DONALD E, 6700 S TOPEKA AVE,66620
296-3782
24 M 1902 55 PH
WILDS, CHARLES E, 3227 MAC VICAR COURT,66611
-
15 M 2834 42 P
WILSON, MARVIN H, 1516 SW SIXTH,66606
234-0591
38 M 1003 64 GS
WINER, RICHARD S, 5813 SW 22ND TERR #4,66614
234-9566
55 M 1902 80 P
WING, NANCY J, 700 HARRISON,66603
295-3175
30 F 3104 56 AOM
WONG, NORMUND, MENNINGER FO,66601
234-9565
34 M 502 59 P
WOOD, EDWARD RUSSELL, 901 GARFIELD,66606
354-9591
49 M 1902 71 IM
WOODS, ROBERT P, 901 GARFIELD,66606
357-6171
14 M 5701 40 N
YORKE JR, CRAIG H, 901 GARFIELD,66606
357-6171
48 M 2401 74 NS
YOUNG, PAUL E, TOPEKA MEDICAL CENTER,66604
233-4927
42 M 2407 75 OPH
YOUNG, THEODORE E, 107 MED ARTS BLDG WEST,66604
232-0576
22 M 2307 46 PD
ZACHARIAS, DAVID LLOYD, 1500 W TENTH,66606
354-6870
26 M 1902 53 PATH
ZIMMERMAN, WILLIAM H, CONTINENTAL BLDG,66606
232-4377
20 M 3006 52 GS

TRIBUNE—316
(Southwest Kansas Society)

WERNER, WILLARD F., ,67879
376-4251
24 M 1902 52 FP

TROY—913
(Northeast Kansas Society)

MASTERSON, MELVIN LEROY, 210 S MAIN,66087
985-2211
23 M 4901 48 R

ULYSSES—316
(Southwest Kansas Society)

BREWER, MARSHALL A, PO BOX 687,67880
356-1261
19 M 1902 46 FP
RAMCHANDANI, A P, 301 E GRANT,67880
356-2432
39 M 49530 74 FP
TILLOTSON, DON R, 223 N MAIN,67880
356-1261
32 M 1902 65 FP

VALLEY CENTER—316
(Sedgwick County Society)

WILSON, ROBERT L, RR 1,67147
685-2563
30 M 1902 57 EM

WAKEENEY—913
(Central Kansas Society)

BERNER, NEAL E, 323 RUSSELL,67672
743-2124
44 M 1902 72 FP
HAMILTON, JAMES J, MEDICAL CENTER,67672
743-2124
30 M 1902 55 FP
JACOBSON, JOEL A, 323 RUSSELL,67672
743-2124
42 M 74811 80 GP

WAMEGO—913
(Pottawatomie County Society)

BORGENDALE, LLEWELLYN V, 1601 SUNSET,66547
456-2291
29 M 1902 60 FP
BRADEN, BILL L, 507 ELM,66547
456-2291
31 M 1902 60 FP
CLARK, LAURENCE A, 507 ELM,66547
456-2291
12 M 1902 42 FP
WELCH, LAUREN A, NATURAL FAMILY PLANNING,66547
275-7913
45 M GS
WIGGLESWORTH, ANNE, HWY 24 PO BOX 149,66606
456-9202
40 F 66547 75 OBG

WASHINGTON—913
(Northeast Kansas Society)

BITZER, DONALD A, 115 W 3RD,66968

03 M 3005 26 DO
HODGSON, DAVID K, 107 E THIRD,66945
325-2259
49 M 1902 78 FP

WATHENA—913
(Northeast Kansas Society)

PETERSON JR, EVAN A, 324 ST JOSEPH ST,66090
989-3122
24 M 1803 55 FP

WELLINGTON—316
(Tri-County Society)

ANDERSON, LARRY R, 1323 NORTH A,67152
326-3301
43 M 1902 73 FP
COLE, WARD M, 110 N JEFFERSON,67152
326-7221
08 M 1902 36 FP
NALDOZA JR, FAUSTINO M, 1323 NORTH A,67152
326-8171
M 74801 64 GS
PEORAZA, HERNANDO, 33 CRESTWAY,67152
326-8070
28 M 26404 56 R
WEIGAND, JOEL T, 1323 NORTH A,67152
326-3301
43 M 1902 70 FP

WESTMORELAND—913
(Pottawatomie County Society)

DECHAIRO, THOMAS, DECHAIRO HOSP,66549
457-3311
13 M 1902 36 FP

WICHITA—316
(Sedgwick County Society)

ABBAS, DILAWER H, 841 S HILLSIDE,67211
685-1111
45 M 70402 71 N
ACEVEDO, ALFREDO, 959 N EMPORIA SUITE 205,67214
265-4701
40 M 73701 59 COTS
AGUSTIN, CONRADO M, 1035 N EMPORIA STE 165,67214
267-3389
38 M 74807 62 OBG
AHLSTRAND, RICHARD A, 3333 E CENTRAL STE 214,67208
685-1291
41 M 3005 67 R
ALDOROTY, NEIL, 3243 E MURDOCK STE 400,67208
686-7351
46 M 64914 75 P
ALEXANDER, ELIZABETH, 855 N HILLSIDE,67214
685-1381
46 F 1902 77 FP
ALFONSO, MANUEL, 3244 E DOUGLAS,67208
689-9445
37 M 84710 66 ANES
ALLEN, PHILLIP M, WESLEY MEDICAL CENTER,67214
688-2838
27 M 2401 54 PATH
ALMONTE, PRISCILLA C, 303 S HILLSIDE,67211
684-7251
44 F 74801 67 ANES
ALMONTE, RODOLFO O, 1431 BLUFFVIEW DR STE 217,67218
686-3791
39 M 74801 64 OBG
AMMAR, ALEX O, 905 N EMPORIA,67214
263-0296
51 M 5101 76 GPVS
ANDERSON, EUGENE G, SUTTON PLACE,67202
265-8619
19 M 1902 44 ORG
ARGOSINO, RODOLFO, 1148 S HILLSIDE,67211
683-5506
40 M 74801 63 GS
ARTZ, TYRONE O, 1125 N TOPEKA,67214
267-0362
41 M 1803 67 ORS
ASHMORE, ARTHUR L, 811 N MISSION,67206
-
05 M 1902 32 TS
AUNINS, JOHN, 4853 HEMLOCK,67216
524-6805
28 M 4706 56 FP
AUSTENFELD, MARK S, 1048 N DEL_ROSE,67208
-
53 M 1902
BAILEY, DONALD C, 3243 E MURDOCK,67208
685-1491
37 M 3901 65 ORS
BARBA, ESTRELLA G, 8309 BROOKHOLLOW LANE,67206
264-2301
41 F 74802 66 P
BARBA JR MD, ANTONIO P, 8309 BROOKHOLLOW LANE,67206
264-2301
34 M 74807 62 OBG

BARKER, BENJAMIN W, 8015 WILLOWBROOK,67207
-
18 M 1902 51 OO
BARNETT, ARNOLD M, 5111 E 21ST,67208
685-2561
32 M 83601 54 N
BARTAL, ELY, 925 N EMPORIA SUITE F,67214
262-7598
45 M 39607 70 ORS
BARTLETT, WAYNE C, 13 HAMPTON RD,67207
689-9124
07 M 1601 31 GS
BASS II, ORAL E, 851 N HILLSIDE,67214
685-1371
40 M 2803 71 U
BATES, MICHAEL D, 2703 EAST CENTRAL,67214
685-1277
48 M 3005 74 OBG
BATTISTE, CYNTHIA ELAINE, 3333 E CENTRAL SUITE 533,67208
681-2021
47 F 1606 73 PD
BAUMAN, M LEON, 1629 UNIVERSITY,67213
-
01 M 1902 44 OO
BAUMANN, PAUL A, 3333 E CENTRAL,67208
685-1291
32 M 5605 57 R
BEAVER, JAMES L, 245 COURTLEIGH DR,67218
253-3262
09 M 2101 35 OO
BEBAK, DONALD M, 2322 E CENTRAL,67214
263-6185
32 M 3508 58 ANES
BECK, CHARLES W, 1431 S BLUFFVIEW STE 111,67218
685-8262
46 M 301 72 IM
BECKER, KARL E, 275 S PERSHING,67218
686-3963
43 M 2307 69 ANES
BENZ, LAURIE J, VETERANS HOSP,67218
685-2221
39 F 4101 65 IM
BETHEL, CHANDLER S, 5107 E 21ST ST,67208
682-6559
34 M 1902 59 IM
BHARGAVA, BAIKUNTH N, 3243 E MURDOCK SUITE 102,67208
682-4523
37 M 49530 63 U
BIERMANN, HENRY J, 425 E MURDOCK,67214
265-6287
27 M 3006 52 GS
BIERMANN, WILLIAM J, 1435 LIEURETT,67203
-
04 M 3006 29 OO
BINGAMAN, ROBERT W, 7111 E 21ST,67206
684-2851
47 M 3901 72 GS
BINYON, KERNIE W, PO BOX 8124 MUNGER STA,67208
684-2819
24 M 1902 56 FP
BLAYLOCK, HOYT C, 835 N HILLSIDE,67214
685-4395
21 M 1902 45 D
BLOOD, MARY J, 547 N HILLSIDE,67214
682-4559
13 F 1902 49 PD
BLOOM, RODNEY LAMONT, 2141 LORI LANE,67207
-
54 M 1902 79 IM
BLOXHAM, THOMAS J, 3244 E DOUGLAS,67208
689-9215
50 M 1803 75 PUD
BOND, ROGER C, 3243 E MURDOCK,67208
684-0251
40 M 5606 67 CD
BOYD, Z REX, 120 S MAIZE RD #12,67209
268-5000
26 M 3005 52 FP
BOYLE, HUGH H, 424 N WOODLAWN,67208
686-2193
33 M 3806 60 PATH
BRAKE, DAVID, 3333 E CENTRAL,67208
685-1291
43 M 702 68 R
BRAUN, KENNETH, 1431 S BLUFFVIEW,67218
683-4688
47 M 3519 72 OPH
BRAUN, THOMAS G, 3243 E MURDOCK #601,67208
685-2377
35 M 6001 61 N
BRAUN III, WILLIAM T, 3333 E CENTRAL,67208
685-1291
37 M 2802 61 R

BRECKBILL, DAVID L, 3333 EAST CENTRAL #214,67208
685-1291
38 M 1902 64 R
BRINTON, E HOLMES, 3244 E DOUGLAS,67208
689-9124
46 M 2101 70 GS
BRINTON, EDWARD S, 329 NORTH TERRACE DR,67208
-
15 M 1511 41 OO
BRITO, RAUL E, 3243 E MURDOCK,67208
682-4523
32 M 31901 59 U
BROSIUS, FRANK C, 3243 E MURDOCK,67208
684-0251
25 M 1902 49 IM
BROWN, DAVID J, 425 EAST MURDOCK,67214
265-6287
45 M 1902 71 GS
BROWN, MICHAEL P, 345 N HILLSIDE,67214
683-6766
51 M 3007 77 OBG
BROWN, ROBERT L, 5025 E KELLOGG,67218
682-1534
21 M 1902 49 FP
BROWN, RONALD C, 3243 E MURDOCK,67208
685-8231
47 M 2803 73 FP
BROWN, RONALD L, 303 S HILLSIDE,67211
684-7251
45 M 3901 71 ANES
BROWN, VAL J, 1802 N HYDRAULIC,67214
265-1461
24 M 1003 47 FP
BROWNING, WILLIAM H, 851 N HILLSIDE,67214
685-1371
16 M 1902 43 U
BRUNER JR, KENNETH W, ST FRANCIS HOSPITAL,67214
268-5414
44 M 2401 70 PATH
BUBECK, RALPH W, 3244 E DOUGLAS,67208
689-9396
36 M 1803 62 IM
BUCK JR, BEN H, 1152 S CLIFTON,67218
684-2081
17 M 2834 43 TS
BUCKRIDAN, LATIF, 7232 CHELSEA,67206
684-7251
31 M 5501 62 ANES
BURNEY, WILLIAM W, 1755 N MADISON,67214
264-8311
17 M 1902 52 FP
BURNEY II, WILLIAM W, 1755 N MADISON,67214
264-8311
50 M 4707 76 IM
BURPEE, JAMES F, 851 N HILLSIDE,67214
685-1371
39 M 5605 66 U
BUTH, DENNIS K, 2916 EAST CENTRAL,67214
684-5243
45 M 1902 72 IM
BUTIN, J WALKER, 3244 E DOUGLAS,67208
689-9477
23 M 1902 47 IM
BUTLER, DORIS C, 1148 S HILLSIDE,67211
684-2329
48 F 1902 75 FP
BYRNE, JAMES PERRY, 905 N EMPORIA,67214
263-0296
42 M 2101 68 TS
CALIENDO JR, DANIEL J, WESLEY MED CENTER,67214
685-2563
41 M 1902 67 EM
CANNON, MICHAEL W, 1035 N EMPORIA,67214
588-6029
50 M 1902 75 JN
CAPPER, STANLEY L, 3244 E DOUGLAS,67208
689-9206
37 M 1803 67 D
CARLSON, TERRY S, 550 N HILLSIDE,67214
688-2820
50 M 3006 77 PATH
CARSON DO, BERTRAM H, ST FRANCIS HOSP,67214
268-5050
25 M 2878 64 EM
CARTER, MACK A, 3333 E CENTRAL,67208
684-5158
18 M 1902 50 OPH
CASEY, JOHN J, 3243 E MURDOCK SUITE 604,67208
685-2377
25 M 2604 49 NS
CAUBLE, WILBUR G, 1148 S HILLSIDE,67211
683-1681
12 M 2834 39 GS

CAWLEY, LED P, WESLEY MED CENTER,67214
685-2151
22 M 3901 52 PATH
CHANG, FREDERIC C, 905 N EMPORIA,67214
263-0296
35 M 2401 59 GS
CHAPMAN, JAMES H, POST OFFICE BOX 11344,67211
265-1584
27 M 4706 63 R
CHARD, FREDERICK H, 3244 E DOUGLAS,67208
689-9129
15 M 5605 39 O
CHI, IL-SUNG, 3333 E CENTRAL SUITE 602,67208
685-4389
41 M 58302 67 ANES
CHO, SECHIN, UKSM - WICHITA,67214
268-8302
47 M 58302 71 PO
CHOPRA, RAMAN, 3333 E CENTRAL #201,67208
685-5271
52 M 49536 76 PD
CHRISTMAN JR, CARL, 550 N LORRAINE,67214
685-0559
48 M 4802 74 OBG
CLARK, COURTNEY, 303 S HILLSIDE,67211
684-7251
30 M 1902 56 ANES
CLEAVER, EDGAR M, 1900 E NINTH,67214
268-8391
26 M 3005 54 PH
CLIFTON, H DAVID, 3600 E HARRY,67218
685-1111
41 M 401 65 R
CLINE, BYRON W, 550 N LORRAINE,67214
685-0559
51 M 4802 77 OBG
COHEN, JUSTIN THOMAS, 3333 E CENTRAL STE 1,67208
684-5158
47 M 2803 74 OPH
COHLMIA, JERRY B, 1035 N EMPORIA,67214
263-5891
43 M 1902 70 IM
COLEMAN, THOMAS J, 959 N EMPORIA,67214
265-0749
18 M 3545 51 IM
COLLIER, HAROLD W, 144 N OLIVER SUITE 325,67208
683-5008
45 M 1902 71 ANES
CONCEPCION JR, EUGENIO S, 1152 SOUTH CLIFTON,67218
681-2401
39 M 74802 64 CD
CONRARDY, PETER A, 1201 N RIVER BLVD,67203
686-7327
42 M 502 69 ANES
COOK, DONALD RAY, 315 N HILLSIDE,67214
686-3391
42 M 2012 71 FP
COOK, G EDWARD, ST JOSEPH HOSPITAL,67218
685-1111
42 M 401 67 R
COOPER, MICHAEL K, 1201 N RIVER BLVD,67218
263-1574
54 M 1902 79 ANES
COSSMAN, F PRICE, 851 N HILLSIDE,67214
685-1371
28 M 1902 57 U
COWLES, GORDON T, 3333 E CENTRAL,67208
683-2661
32 M 1902 58 OBG
CRANE, DAVID D, 929 N ST FRANCIS,67214
262-6211
34 M 2501 60 PATH
CRONIN, DONALD J, 3244 E DOUGLAS,67208
689-9227
16 M 2604 40 ENT
CROW, ERNEST W, 3243 E MURDOCK,67208
684-0252
20 M 1902 44 CD
CROWLEY, EDWARD X, 345 N HILLSIDE,67214
682-4519
14 M 1543 39 GYN
CUMMINGS, RICHARD J, 427 N HILLSIDE,67214
686-6608
32 M 1902 57 OTO
DAKHIL, SHAKER R, 1035 N EMPORIA SUITE 210,67214
262-4467
50 M 60501 75 IM
DANIELS, ROBERT M, 929 N ST FRANCIS,67214
262-6211
24 M 1902 54 FP
DANIELS III, H BRUCE, 1122 N TOPEKA,67214
-
55 M 4802 81 FP

DARRAH, CARDL J N, 3333 E CENTRAL SUITE 613,67208
685-9289
49 F 1902 74 R
DAVIDSON, HARRY T, 556 N BROADVIEW,67208
-
87 M 3802 11 OO
DAVIS, PAUL H, 7111 E 21ST,67206
684-2851
47 M 3901 72 FP
DAVIS, RONALD B, 1148 S HILLSIDE,67211
685-2152
45 M 1902 72 FP
DAY, HOWARD, 1035 N EMPORIA -SUITE 235,67214
263-5891
48 M 1902 74 NEP
DE BAKKER, JAN B, 1035 N EMPORIA,67214
263-4903
25 M 5104 59 GS
DE HART, ARTHUR DONIVA, 2703 E CENTRAL,67214
685-1277
50 M 4804 77 OBG
DEJONG, DAVID C, 550 WEST SHORE DR,67209
268-5424
33 M 2501 59 PATH
DEMOSS, ELEANOR P, 3333 E CENTRAL SUITE 407,67208
682-5591
42 F 74802 66 PO
DEPOE, JOSEPH H, 8608 ENT DR,67210
221-4980
31 M 1902 57 FP
DIACON, JAMES L, 2029 N WOODLAWN #522,67208
326-2111
24 M 3901 52 FP
DIRKSEN, HANS C, 550 N HILLSIDE,67214
688-2360
47 M 6001 71 NEU
DOEBLIN, P LAURENCE, 3333 E CENTRAL SUITE 214,67208
685-1291
40 M 1002 73 R
DOLAN JR, PHILIP JARVIS, 3244 E DOUGLAS,67208
689-9477
47 M 2101 73 GE
DONATELLE, EDWARD P, UKSM WICHITA,67214
268-8221
22 M 2604 50 FP
DONLEY, JAMES L, 3101 E NINTH,67208
684-0208
46 M 1902 72 P
DONNELL, JAMES M, 2320 E CENTRAL,67214
682-6121
28 M 1902 55 FP
DOORNBOS, J FRED, 929 N ST FRANCIS,67214
262-6211
28 M 1902 57 R
DOWNING, GREGORY, 1123 NORTH OLIVER,67208
685-8499
M 1902
DRAKE, RALPH L, 4422 E 3RD,67208
-
99 M 4102 26 OO
DRANGE, ROBERT K, 3333 E CENTRAL STE 214,67208
685-1291
52 M 1902 77 OR
DRAZEK, GEORGE, 3244 E DOUGLAS,67208
689-9111
50 M 3506 76 OPH
DREVETS, CURTIS C, 3244 E DOUGLAS,67208
689-9178
30 M 1902 56 IM
DUICK, GREGORY, 1035 NORTH EMPORIA #130,67214
263-3271
46 M 1543 72 CD
DURAND, ANTONIO C, 959 N EMPORIA,67214
263-7893
29 M 74807 56 U
DYCK, CORA E, 702 #AVERLY,67218
-
00 F 1902 26 OO
DYER, VERNON E, 3244 EAST DOUGLAS,67208
262-6202
36 M 301 72 OBG
ECKERT, WILLIAM G, ST FRANCIS LAB,67214
262-6211
26 M 3519 52 PATH
EDWARDS, MANIS C, 3333 E CENTRAL,67208
683-2661
33 M 3005 58 OBG
EGELHOF, RICHARD H, 1035 N EMPORIA SUITE 290,67214
263-1299
45 M 1902 73 FP
ELLIS, HARVEY D, 6611 E CENTRAL,67206
683-1022
24 M 1902 55 GS

ELNEN, WALTER T, 460 N TERR DR,67208
682-5571
03 M 1643 32 GS
ENOCH, ROLLAND, 315 N HILLSIDE,67214
681-0423
49 M 64914 76 FP
EPLEE, JOHN P, ST JOSEPH MED CENTER,67218
685-1111
53 M
ERKEN, RONALD V, WICHITA PSYCHIATRIC CTR,67208
684-0201
29 M 2834 56 P
ESTEP, THOMAS H, 905 N EMPORIA,67214
263-0295
51 M 6002 75 CD
EVANS, FARRIS O, 521 RUTLAND RD,67206
-
05 M 1902 32 FP
EVANS, GRANT E, 4618 E CENTRAL,67208
682-6556
21 M 4901 46 FP
EVANS, JOHN F, 8542 MT VERNON CT,67207
688-2360
42 M 2803 70 MFM
EVANS, ROGER WILLIAMS, 933 N TOPEKA,67214
263-5889
39 M 1902 64 CD
EYSTER, ROBERT L, 3243 E MURDOCK,67208
685-1491
47 M 3901 73 ORS
FARHA, GEORGE J, 905 N EMPORIA,67214
263-0296
27 M 2101 57 GS
FARHA, S JIM, 905 N EMPORIA,67214
263-0295
31 M 1001 57 TS
FENDER JR, THOMAS H, 9400 E LINCOLN APT 113,67207
267-8439
25 M 4812 54 OS
FERRELL, DONALD P, ST JOSEPH MEDICAL CTR,67218
685-2371
36 M 3901 63 EM
FERRIS, BRUCE G, 825 N EMPORIA,67214
262-3495
43 M 1902 69 PS
FEUILLE JR, EDMOND G, 212 N HILLSIDE,67214
682-4572
50 M 4802 75 OBG
FIELDS DO, STEPHEN A, 7200 W 13TH,67212
722-4258
42 M 2878 72 FP
FINLEY, DENNIS R, 1035 N EMPORIA,67214
262-7429
36 M 1506 62 ORS
FISHER, JAMES B, 141 S OLD MANOR,67218
-
09 M 1902 36 OO
FISHER, RAY F, 3243 E MURDOCK SUITE 500,67208
684-0251
49 M 1902 74 IM
FITZGERALD, EDWARD J, 3600 E HARRY,67218
685-1111
22 M 3006 50 R
FITZIG, SANFORD, 3244 E DOUGLAS,67208
689-9344
45 M 4102 72 U
FLEMING, FORNEY W, 3243 E MURDOCK #200,67208
685-1491
43 M 4802 69 ORS
FLOWERS JR, CLELL B, 855 N HILLSIDE,67214
685-1381
22 M 1902 55 FP
FORD, CHARLES R, 232 S MAIZE RD,67209
942-4279
38 M 1902 63 OPH
FOWLER, ROBERT J, 3244 E DOUGLAS,67208
689-9236
37 M 2802 63 IM
FRANCIS, NORTON L, 55 VIA ROMA,67230
-
10 M 3005 35 ENT
FRANCISCO, DAN A, 3243 MURDOCK SUITE 500,67208
684-0251
40 M 1803 75 CO
FRANCISCO, LINDA L, 3243 E MURDOCK SUITE 500,67208
684-0251
47 F 1803 74 NEP
FRENCH, JAMES E, 2240 CAMERON CIRCLE,67226
-
53 M 3005 78 GS
FRENCH, JEROME E, 5205 E 21ST,67208
684-2838
44 M 1103 71 OTO

FRITZ, GEORGE E, WESLEY MED CENTER,67214
685-2151
18 M 2507 43 PATH
FRITZEMEIER, WILLIAM H, 835 N HILLSIDE,67214
685-4395
14 M 1902 41 D
FROMER, JOEL, 2627 E CENTRAL,67214
684-0501
46 M 16501 75 A
FROMM, ARTHUR H, 315 N HILLSIDE,67214
685-2281
37 M 1902 63 FP
FULTON, JOHN K, 3333 E CENTRAL SUITE 801,67208
686-0732
18 M 5605 43 PUO
GALICHIA, JOSEPH P, PO BOX 594,67201
263-3271
42 M 1902 69 CD
GALVAN, ALONSO, 3243 E MURDOCK,67208
684-0251
35 M 64906 64 IM
GENILO, AMANCIO C, 120 LONGFORD CT,67206
685-2371
37 M 74801 61 EM
GENILO, CELESTE A, 3244 EAST DOUGLAS,67208
689-9445
39 F 74801 62 ANES
GEORGE, EARL F, 5107 E 21ST,67208
265-6991
35 M 1902 65 FP
GEORGE, M DON, WICHITA PSYCHIATRIC CL,67208
268-8388
31 M 1001 56 P
GERBER, ALLEN D, 7111 E 21ST,67206
684-2851
48 M 1902 71 GS
GESSLER, DONALD J, 1122 N TOPEKA,67214
265-2876
41 M 1902 67 FP
GILMARTIN, RICHARD C, 3243 E MURDOCK #601,67208
685-2377
32 M 4101 58 PDN
GIVNER, DAVID, 611 N HILLSIDE,67214
684-0501
03 M 2301 29 IM
GOHIL, MAHENDRA N, 1823 RUTLAND,67206
265-1684
28 M 40930 66 DR
GOLDBERG, HERBERT R, 400 WOODLAWN SUITE 101,67208
685-4215
33 M 3508 59 PD
GONZALEZ, HIRAM, 1035 N EMPORIA,67214
262-1853
20 M 54901 52 P
GOODPASTURE, HEWITT C, 3333 E CENTRAL SUITE 410,67208
682-7371
43 M 1902 69 IM
GOULDNER, RENE M, 139 COURTLEIGH DR,67218
-
88 M 3501 18 OO
GOYLE, KRISHAN K, 1144 N ST FRANCIS,67214
267-0159
34 M 49529 63 CD
GOYLE, VIMAL, 1150 N ST FRANCIS,67214
257-9906
41 F 49529 65 OBG
GRAUEL, CHARLES W, 8310 CHALET,67207
262-1127
44 M 1902 70 ANES
GRAVES, JACK W, 3244 E DOUGLAS,67208
-
17 M 1902 42 OO
GRAY, C LUCIEN, 4821 E CENTRAL,67208
684-5171
21 M 1902 45 ENT
GRAY, H TOM, 3244 E DOUGLAS,67208
689-9152
19 M 401 44 D
GREER, JAMES A, 3244 EAST DOUGLAS,67208
689-9227
43 M 1611 69 OTO
GRIBBLE, ROBERT N, 1035 N EMPORIA #285,67214
264-2401
43 M 1902 69 R
GRILLOT, FLOYD B, 814 S WOODLAWN,67218
684-0243
18 M 1902 51 FP
GROHS, HEINZ K, 550 NORTH HILLSIDE,67214
685-2151
42 M 15407 66 PATH
GRUNO, FRANK M, 3244 EAST DOUGLAS,67208
689-9420
47 M 1803 72 IM

GRUSHNYS, ARNOLD, 3244 E DOUGLAS,67208
689-944S
19 M 40721 59 ANES
GSELL, GEORGE F, 32 MISSION RD,67206
-
07 M 1601 33 OO
GUTHRIE, RICHARD A, 1001 N MINNEAPOLIS,67214
268-8228
3S M 2803 60 PD
GUTTIKONDA, PRASAD B, UKSM WICHITA,67214
-
45 M 49557 72 P
HABASHY, SHAWKY N F, 1122 N TOPEKA,67214
268-5996
43 M 33004 65 OBG
HAGAN, C THOMAS, 9S9 N EMPORIA,67214
265-0789
16 M 3006 42 IM
HAGAN, FRANCIS J, PO BOX 1837,67201
262-1057
13 M 3006 39 FP
HALE, RALPH, 847 S HILLSIDE,67211
684-0295
18 M 1902 46 A
HALL, J ROGER, 3333 E CENTRAL,67208
685-S227
42 M 4802 68 OPH
HALPIN, EDWARD D, 1148 S HILLSIDE,67211
685-9229
33 M 1902 62 OBG
HARMS, EDWIN M, 5623 POLO DR,67208
-
06 M 3901 34 OO
HARRIS, FRANK H, 1035 N EMPORIA,67214
-
09 M 1001 39 NP
HARRISON, A BROOKS, 1040 STRATFORD,67206
-
99 M 1902 25 OO
HARRISON, PAUL BARRY, 3243 EAST MURDOCK,67208
685-6222
49 M 1902 74 GS
HART, DILLIS L, 1152 S CLIFTON,67218
684-2081
35 M 3901 64 GS
HARTLEY, JAMES M, UKSM - WICHITA,67214
685-2387
45 M 2604 71 FP
HARVEY, ROSEMARY B, 635 N MAIN,67203
265-5674
24 F 1902 49 ADM
HASSAN, RIZWAN U, 841 S HILLSIDE,67211
685-2831
47 M 70305 70 N
HATTRUP, RICHARD J, 610 N TYLER RD,67212
681-3016
31 M 3006 57 FP
HAWLEY, RAYMOND G, ST JOSEPH HOSP,67218
685-1111
39 M 1902 65 PATH
HAYES, KRIS A, 649 N DELLROSE,67208
257-S971

HAYES, WILLIAM L, 3243 E MURDOCK,67208
684-0251
28 M 1902 53 CD
HAYS, THOMAS H, 7111 E 21ST,67206
684-2851
49 M 1902 75 FP
HENNING, CHARLES E, 320 N HILLSIDE,67214
682-3221
37 M 1902 63 ORS
HENSLEY JR, CLINE D, 3244 E DOUGLAS,67208
689-9272
20 M 1902 44 ORS
HERED, JOHN, 959 N EMPORIA SUITE 403,67214
262-3613
41 M 2802 67 N
HERSHBERGER DO, GROVER, 1245 NW ST,67203
945-6910
47 M 2878 79 GP
HERSHORN, SIMON E, 3333 E CENTRAL,67208
685-1291
22 M 1902 46 R
HIEBERT, ABRAHAM E, 1530 W 13,67203
-
94 M 2802 25 OO
HINSHAW, ALFRED H, 6110 ONEIDA,67208
-
07 M 1902 33 OO
HINSHAW, CHARLES T, 256 N BLECKLEY DR,67208
-
99 M 4705 26 OO

HINSHAW JR, CHARLES T, 371S E DOUGLAS,67218
684-7784
32 M 1902 58 PATH
HIRATZKA, TOMIHARU, 6 ST CLOUD PLACE,67230
-
13 M S11 43 OO
HIZON, RAMON R, ST FRANCIS HOSP,67214
262-6211
38 M 74801 62 OR
HODGSON, ROBERT B, 841 NORTH BROADWAY,67214
253-5131
22 M 1902 47 FP
HODSON, HERVEY R, 1122 S CLIFTON,67218
-
03 M 1606 31 OO
HOLDEN, DAVID M, 3243 E MURDOCK SUITE 300,67208
688-3070
38 M 801 63 FP
HOLDEN JR, RAYMOND F, 262 SOUTH BROOKSIDE,67218
-
10 M 2802 33 OO
HOLLADAY, HARMON M, 327 N RUTAN,67208
681-0431
23 M 1902 52 ANES
HOLMES, JED, 7111 E 21ST,67206
684-2851
53 M 3005 78 FP
HORBELT, DOUGLAS V, 3333 E CENTRAL,67208
682-6511
47 M 4802 72 OBG
HOUSHOLDER, DANIEL FAIR, 7705 KILLARNEY CT,67206
268-5910
43 M 1902 70 NM
HOUSHOLDER, MARTHA S, 835 NORTH HILLSIDE,67214
68S-439S
46 F 1902 72 D
HOWARD, DONALD O, 9S9 N EMPORIA,67214
265-7241
11 M 1902 38 OPH
HUEBERT, DEAN A, 5025 E KELLOGG,67218
682-1534
22 M 1902 46 FP
HULL, KENNETH L, 3429 E DOUGLAS,67218
265-7903
38 M 2301 69 P
HULTGREN, MYRON K, 1520 S CLIFTON,67218
685-1381
41 M 1902 68 FP
HUME, JOSEPH W, 5111 E 21ST,67208
685-2223
38 M 1902 69 OBG
HUMMER, LLOYD M, 3244 E DOUGLAS,67208
689-9323
32 M 3901 57 IM
HUND, LARRY R, 3333 E CENTRAL,67208
363-84S9
52 M 1902 78 PD
HUSTEAD, ROBERT F, 427 N HILLSIDE,67214
683-7200
28 M 801 54 ANES
HYNES, HARRY E, 103S N EMPORIA,67214
262-4467
35 M 53902 58 HEM
IBARRA, J LUIS, 1035 N EMPORIA,67214
262-1853
20 M 64901 46 P
IDBEIS, BADR, 1144 N ST FRANCIS,67214
267-0159
47 M 87501 72 TS
IOANNOU, NICHOLOAS, 1111 N ST FRANCIS,67214
265-2613
48 M 41801 72 CDTS
JACKSON, CATHERINE M, UKSM WICHITA,67214
268-8388
40 F 35207 64 CHP
JACKSON, CHARLES R, 1035 N EMPORIA,67214
263-0812
27 M 1606 53 GS
JAMES, DONALD L, 1301 N WEST,67203
945-S245
42 M 3901 71 OTO
JAMES, VERNON L, 3333 E CENTRAL SUITE 816,67208
685-5326
29 M 3501 55 PD
JAWADI, JAMEELA HUSAIN, 3333 E CENTRAL SUITE 610,67226
685-6659
50 F 49521 73 PD
JAZAYERLI, NABIL, 1152 S CLIFTON,67218
681-2401
44 M 87501 70 CD
JEHAN, SAYED S, 635 N MAIN,67202
268-8306
33 M 70403 59 P

JENNEY, CHARLES B, 905 N EMPORIA,67214
263-0296
34 M 2834 61 GS
JESTER, SHELBY L, 250 N PERSHING,67208
263-1574
43 F 4102 74 ANES
JOHNSON, CAROL ANN, 3243 E MURDOCK SUITE 303,67208
688-3070
49 F 1902 77 FP
JOHNSON, GEORGE K, UKSM WICHITA,67218
268-5984
40 M 1205 67 IM
JOHNSON, THOMAS E, 3333 E CENTRAL,67208
685-1291
41 M 1643 67 R
JONES, JAY S, 1812 LONGFORD CT,67207
-
50 M 64914 77 ORS
JUDILLA JR, FRANCISCO, 2322 EAST CENTRAL,67214
263-6186
44 M 74801 71 ANES
KADISON, HERBERT I, ST FRANCIS HOSP,67214
262-6211
44 M 1511 69 R
KARDATZKE, E STANLEY, 10300 W MAPLE,67209
943-9342
39 M 1720 64 FP
KARDATZKE, JON K, 10300 MAPLE,67209
943-3271
36 M 1720 62 FP
KASHA, ROBERT L, 5025 E KELLOGG,67218
682-1534
11 M 2834 38 GS
KASSEBAUM, KENNETH G, 3243 E MURDOCK SUITE 400,67208
685-5381
34 M 1606 60 CHP
KAUFMAN, EUGENE E, 3243 E MURDOCK,67208
685-1491
30 M 1902 56 ORS
KEENE, GEORGE H, 5025 E KELLOGG,67218
682-1534
20 M 1902 49 GS
KELLER, JAMES P, 1431 S BLUFFVIEW STE 209,67218
685-1284
48 M 1902 74 IM
KELLERMAN, RICK, 3243 E MURDOCK STE 303,67208
688-3070
54 M 1902 78 FP
KELLY, ROBERT W, 550 N LORRAINE,67214
682-6511
46 M 4802 72 OBG
KENDALL, TOM E, 825 N EMPORIA,67214
262-3495
37 M 3901 62 PS
KENDRICK, J GILLERAN, 3333 E CENTRAL,67208
682-5511
20 M 1902 46 OBG
KENNEDY, GERALD T, 16069 CAVALCADE LANE,67230
684-5243
35 M 1902 61 GE
KHICHA, GYANCHAND J, 905 N EMPORIA,67214
263-0296
37 M 49530 61 TS
KHOURY, GEORGE H, 3333 E CENTRAL,67208
681-2021
32 M 33002 55 PD
KIM, PAIK N, 1035 N EMPORIA,67214
262-4467
33 M 58302 58 HEM
KIMBLE, JAMES A, 3244 E DOUGLAS,67208
689-9316
45 M 702 71 OPH
KIRK JR, E DAVID, 1431 S BLUFFVIEW DRIVE,67218
685-1351
34 M 1902 62 IM
KISER, JOHN L, 3243 E MURDOCK,67208
685-6222
37 M 2802 62 GS
KISER, WILLARD J, 3243 E MURDOCK,67208
-
05 M 4705 30 GS
KITCHEN, ROBERT R, 3420 E DOUGLAS,67208
685-2355
26 M 1902 52 CHP
KLONTZ, WILLIAM JOSEPH, UKSM WICHITA,67214
268-8388
51 M 1902 76 P
KNAPP, LESLIE E, 302 S CRESTWAY,67218
-
95 M 1902 25 OO
KNAPP, M ROBERT, 37 VIA ROMA,67230
684-7251
23 M 3519 47 ANES
KNEIDEL, THOMAS W, 732 N TOPEKA,67214
267-1924
40 M 4101 66 ORS
KNIGHT, PHILIP J, 905 N EMPORIA,67214
263-0296
42 M 0502 68 PDS
KOURI, SAMMY H, 3243 E MURDOCK,67208
682-2911
33 M 3901 57 GS
KRAUSE, ROLAND L, 855 N HILLSIDE,67214
685-1381
25 M 1902 53 IM
KRUPKA, JOHN J, 959 N EMPORIA SUITE 403,67214
262-3513
47 M 1642 73 NS
KUBINA, GLENN RICHARD, 5205 E 21ST,67208
684-2838
47 M 3840 72 OTO
KURTH, C JOSEPH, 925 N EMPORIA,67214
263-6177
10 M 3006 35 NP
KUTILEK, FRANK J, 7200 W 13TH ST SUITE 3,67212
722-4258
30 M 1902 57 FP
LACEY, STEFFAN R, ST FRANCIS HOSP,67214
268-5470
50 M 3005 76 PATH
LAI, JENG Y, 959 N EMPORIA STE 205,67214
265-2613
41 M 38502 67 TS
LANCE JR, JOHN F, 3244 E DOUGLAS,67208
689-9438
20 M 1902 45 ORS
LATIMER, KATHERINE, 3243 E MURDOCK SUITE 401,67208
686-7327
49 F 1205 75 ANES
LAUVER, MARY ANN, 1001 N MINNEAPOLIS,67214
268-8302
40 F 1902 74 PD
LAWN, CLAUDIA A, 1035 N EMPORIA SUITE 285,67214
265-1684
50 F 1902 75 R
LAWN, RAYMOND A, 715 N MISSION RD,67206
683-8991
09 M 2504 35 AM
LAZAR, HARRY, 611 N HILLSIDE,67214
-
08 M 1611 35 A
LEE, R REX, 5155 E HARRY,67218
685-2306
29 M 3901 55 FP
LEE JR, EDWARD S, 2002 E 17TH ST,67214
-
09 M 4707 37 FP
LEISY, JERALD W, 3310 E DOUGLAS SUITE 101,67208
681-2937
42 M 1902 68 P
LEVINE, WILLIAM R, UKSM-WICHITA,67214
268-8388
42 M 1902 67 P
LEWIS, LARRY D, 2255 S BROADVIEW,67218
-
54 M 3901 80 FP
LIES, RICHARD B, 3244 E DOUGLAS,67208
689-9111
42 M 1902 68 RHU
LIN, JOE J, 8630 HUNTINGTON,67206
268-5420
42 M 24404 69 PATH
LINHARDT, RONALD D, 3243 E MURDOCK,67208
533-2655
36 M 2803 64 OBG
LITTELL, JAMES A, 1520 S CLIFTON,67218
685-2371
44 M 1902 71 EM
LITTLE, L GILBERT, 122 S WESTFIELD AVE,67209
-
99 M 401 27 P
LOCKHART, JOSEPH G, 5900 E CENTRAL,67208
684-7239
17 M 4113 43 PD
LOEFFLER, JAMES A, 400 N WOODLAWN,67208
685-5375
36 M 3841 63 A
LOEWEN, HENRY H, 2142 W 17TH,67203
-
03 M 1902 35 OO
LOEWEN, WILLIAM C, 10300 W MAPLE,67209
722-6892
41 M 1902 71 FP
LOGAN, GEOFFREY G, 212 N HILLSIDE,67214
682-4572
31 M 14303 56 OBG

LOSEE, JOHN M, 3244 E MURDOCK STE 401,67208
686-7327
51 M 4301 77 ANES
LOVETT, PAUL A, 110 PATTON,67208
-
09 M 1902 45 ORS
LOW, HAROLD L, 1148 S HILLSIDE,67211
684-2858
18 M 1902 44 FP
LUEKEN, LUEKE B, 3244 E DOUGLAS,67208
689-9234
23 M 40723 52 OBG
LUELLEN, THOMAS J, 3244 E DOUGLAS,67208
689-9244
17 M 1902 41 IM
LUZZATI, ENZO F, ST FRANCIS HOSPITAL,67214
262-6211
25 M 56119 50 R
LYNCH, MARY A, 320 N HILLSIDE,67214
682-3221
48 F 1002 77 SM
MADISON JR, WARD N, 3600 E HARRY,67218
686-9432
37 M 3601 62 PATH
MAGIDSON, ELLIOT ARTHUR, 116 LONGFORD CT,67206
686-2193
43 M 1611 68 PATH
MAILMAN, GERSHOM, ST JOSEPH MED CENTER,67218
685-1111
26 M 2803 49 ANES
MALIK, MUMTAZ ILAHI, 1035 N EMPORIA 290,67214
265-1631
47 M 70401 70 ORS
MANNING, ROBERT T, 1001 N MINNEAPOLIS,67214
268-8378
27 M 1902 54 IM
MANSOUR, BADIE S, 3243 E MURDOCK SUITE 401,67208
686-7327
45 M 33002 69 ANES
MARSH, HENRY O, 925 N EMPORIA,67214
262-7598
18 M 1611 43 ORS
MARTIN JR, GLEN E, 2322 E CENTRAL,67214
263-6186
20 M 1902 49 ANES
MARYMONT JR, JESSE H, WESLEY MED CENTER,67214
685-2151
28 M 3515 54 PATH
MASSEY, ANDREW D, 1001 N MINNEAPOLIS,67214
268-8378
50 M 1902 77 IM
MASTIO JR, GEORGE J, 3243 E MURDOCK,67208
684-5235
25 M 1902 52 GS
MATASSARIN, BENJAMIN M, 2916 E CENTRAL,67214
684-5243
20 M 1902 45 IM
MATASSARIN, FREDERICK W, 734 N EMPORIA,67214
265-2382
15 M 1902 37 U
MAWDSLEY, MICHAEL W, 3333 EAST CENTRAL #610,67208
686-6659
49 M 1902 74 PD
MCBRIDE, ANN, UKSM-WICHITA,67214
268-8378
51 F 1902 76 IM
MCCLANAHAN, WARD A, 5105 E 21ST,67208
684-8211
22 M 3005 48 ORS
MCCLELLAN, ERNEST L, 3243 E MURDOCK SUITE 401,67208
686-7327
38 M 4802 70 ANES
MCCOY, CHARLES P, 3333 E CENTRAL,67208
682-6511
17 M 3006 42 OBG
MCGUIRE, WILLIAM F, 3333 E CENTRAL,67208
683-5655
17 M 4101 43 PD
MCMULLEN, BRUCE R, 1122 S CLIFTON,67218
682-5012
53 M 4002 79 IM
MCNICKLE, GEORGE ANDREW, 222 S RIDGE RD, 67209
945-0142
47 M 1902 75 FP
MCQUEEN, DAVID ARNOLD, 818 N EMPORIA,67214
262-7598
47 M 64914 75 ORS
MCREYNOLDS, CHARLES R, WESLEY MED CENTER,67214
688-2899
21 M 3840 47 PATH
MEADOWS, DONALD C, 3333 E CENTRAL,67208
683-5611
36 M 102 69 OPH

MEEKER II, BRUCE P, 345 N HILLSIDE,67214
686-3384
30 M 1902 58 OBG
MELEAN, JAIME, 1152 SOUTH CLIFTON,67218
681-2401
40 M 17602 65 CD
MELHORN, J MARK, 656 S LORRAINE,67211
268-5989
53 M 1902 80 ORS
MELHORN MD, KATHERINE J, 656 S LORRAINE,67211
268-8302
55 F 1902 81 PD
MENAKER, JEROME S, 2703 E CENTRAL,67214
685-1277
16 M 1002 41 OBG
MENDIONES, L MARLENE, 835 N HILLSIDE,67214
685-4395
45 F 1611 70 D
MENDIONES, RUPERTO D, 3243 E MURDOCK -SUITE 404,67208
682-5585
44 M 1611 71 IM
MENEHAN, H JAMES, 3244 E DOUGLAS,67208
689-9404
26 M 1902 53 PD
MENKING, F W MANFRED, 3244 E DOUGLAS,67208
689-9336
34 M 40715 61 PD
MENKING, SUSAN MARGARET, UKSM WICHITA,67214
268-8302
41 F 3840 67 PD
MERCADER, MARIO S, 2322 E CENTRAL,67214
263-6185
43 M 74801 64 ANES
MEREDITH, W TOM, 1035 N EMPORIA,67214
263-7285
35 M 4812 61 IM
MERRITT, JOE P, 3243 E MURDOCK,67208
688-5211
46 M 2802 71 OBG
MERSHON, JAMES C, 933 N TOPEKA,67214
263-5889
37 M 1803 63 IM
MEYER, WARREN E, 1144 S CLIFTON,67218
684-5237
27 M 1606 51 GS
MICHELBACH, ALBERT P, 2916 E CENTRAL,67214
684-5243
35 M 2101 61 IM
MILFELD, DOUGLAS J, 905 N EMPORIA,67214
263-0296
45 M 4804 72 TS
MILLER, CLYDE W, 7618 DONEGAL,67206
-
09 M 2002 36 OD
MILLER, DAVID PATERSON, 7111 E 21ST N,67206
688-3070
50 M 2803 77 FP
MILLER, DON E, 4145 E KELLOGG,67218
682-6551
20 M 2802 44 GS
MILLER, PHILIP A, 4 PEACHTREE LN,67207
684-2081
44 M 1902 70 GS
MILLS, CHARLES D, 1140 S WATER,67213
-
89 M 2002 14 OD
MILLS, GEORGE QUINTON, 550 N HILLSIDE,67214
688-2810
48 M 3006 73 PATH
MINNS, GAROLD D, UKSM-WICHITA DEPT OF MED,67214
268-8378
51 M 1902 76 IM
MIRZA, MEDO, 3333 E CENTRAL,67208
686-6683
38 M 40733 64 PDS
MOHR, SANDRA NELL, 400 W CENTRAL #3105,67203
-
57 F 1902
MOORE, DENNIS F, 1035 N EMPORIA,67214
265-3226
36 M 2101 62 HEM
MOORE, JANE A, WESLEY MED CENTER,67214
688-2239
50 F 3901 76 EM
MORGAN, DICK A, 3243 E MURDOCK SUITE 401,67208
686-7327
43 M 3901 69 ANES
MORGAN, JAMES I, 3124 S SENECA,67217
522-2266
29 M 1606 53 FP
MORGAN III, LOUIS S, 8030 E KELLOGG,67207
683-3811
22 M 3901 48 FP

MORRISON, RICHARD L, 1148 S HILLSIDE,67211
564-2423
42 M 1902 67 FP
MORROW, THOMAS F, 3310 E DOUGALS,67208
685-1443
21 M S606 46 P
MOSELEY, JACK E, 1120 S CLIFTON,67218
682-4982
25 M 3901 53 FP
MOSIER, STANLEY JAY, 3243 E MURDOCK,67208
685-8231
42 M 1902 68 FP
MUELLER, VERNETTE A, 1431 S BLUFFVIEW,67218
684-3981
17 M 2802 41 DBG
MUETH, JOAN D, 7111 E 21ST,67206
684-2858
53 F 2803 79 FP
MULLINIX, JANICE M, 3243 MURDOCK SUITE 601,67208
685-2377
47 F 3006 73 N
MURPHY, BARRY L, 3243 EAST MURDOCK #500,67208
684-0251
45 M 1902 71 IM
MURPHY, DUANE A, 3243 E MURDOCK,67208
685-1491
32 M 1902 65 ORS
MURPHY, PAUL M, 3600 E HARRY,67218
685-1111
28 M 3006 51 R
MURRAY, KENT B, VETERANS MEDICAL CENTER,67218
263-6131
47 M 3901 73 IM
NELSON, GERALD D, 825 N EMPORIA,67214
262-3495
34 M 1902 60 DS
NELSON, RUSSELL ALAN, 3333 E CENTRAL,67208
685-5271
18 M 1902 45 PD
NELSON JR, GUST H, 3600 E HARRY,67218
685-1111
23 M 1902 46 DR
NESMITH, LESLIE W, 3333 E CENTRAL,67208
683-5611
40 M 1902 66 OPH
NEWBY, JAMES P, 905 N EMPORIA,67214
263-0296
34 M 1902 59 TS
NEWSOM, F CARTER, 3310 E DOUGLAS,67208
685-1443
19 M 1201 43 P
NIXON, WILLIAM A, 3333 E CENTRAL SUITE 525,67208
683-6622
16 M 1902 44 GS
NORRIS, ROBERT P, 3244 E DOUGLAS,67208
689-9232
17 M 1902 43 IM
NORTH, DORIS G, 1148 S HILLSIDE,67211
684-5257
16 F 1902 47 FP
NORTH, VICTOR, 1148 S HILLSIDE,67211
684-5257
18 M 1902 47 FP
NORTON, ROBERT K, 3244 E DOUGLAS,67208
689-9235
32 M 1001 57 DD
NOWLIN, NANCY S, 1001 N MINNEAPOLIS,67214
268-8378
47 F 1902 74 IM
NUILA, RICHARD F, 3244 E DOUGLAS,67208
639-9453
50 M 34104 76 OBG
NYBERG, FREDRIK F, 3333 E CENTRAL,67208
685-7234
22 M 2101 46 GYN
O'DONNELL JR, LEONARD A, 8033 E DOUGLAS,67207
684-2835
27 M 1902 55 IM
O'DONNELL SR, LEONARD A, 7 ST JAMES PLACE,67206
-
02 M 5606 26 OO
OCHSNER, BRUCE B, 1035 N EMPORIA STE 215,67214
253-6273
39 M 1902 65 OPH
ODENHEIMER, BURTRAM J, 3244 E DOUGLAS,67208
689-9137
48 M 2105 73 N
OLSON, BARBARA J, 3244 E DOUGLAS,67208
689-9137
49 F 5605 76 CHN
OSBORNE, CONRAD C, 855 N HILLSIDE,67214
685-1381
38 M 1902 67 FP

OSIO, ANTONIO L, 1520 S CLIFTON,67218
685-2371
41 M 26404 65 EM
OSOBA, WILLIAM G, 2525 W 13TH,67203
943-9391
25 M 2802 51 FP
OWEN, LARUE W, 303 S HILLSIDE,67211
684-7251
19 M 1902 50 ANES
OWEN, PERE A, 1128 S CLIFTON,67218
684-7251
37 M 1902 64 ANES
PADGETT, WILLIAM L, 606 STRATFORD,67206
-
24 M 1902 51 FP
PAGE, RUTH, 1051 N STRATFORD,67206
-
13 F 1902 43 OO
PALMER, DAVID L, 4805 W CENTRAL,67212
945-5177
37 M 1902 63 A
PARK, ROGER WALTER, 3244 E DOUGLAS,67208
689-9217
43 M 1902 69 DD
PARKER, HAROLD L, 2932 BENJAMIN,67204
262-3765
32 M 1902 67 FP
PATTERSON, BRUCE W, 1520 SOUTH CLIFTON,67218
685-2371
46 M 1902 73 EM
PATTON, J MICHAEL, 1520 S CLIFTON,67207
685-2371
51 M 3005 78 FP
PAY, NORMAN T, ST FRANCIS HOSP,67214
268-5914
45 M 74802 68 NR
PENCE, CHARLES D, 3244 EAST DOUGLAS,67208
689-9468
42 M 1902 68 ORS
PENNINGTON, KATHERINE, 2113 SO BLUFF CT,67218
685-5271
16 F 1902 43 PD
PETERIE, JERRY P, 3333 E CENTRAL SUITE 410,67208
682-7371
48 M 1902 75 IM
PETERS, THOMAS J, 3244 E DOUGLAS,67208
689-9190
47 M 2803 77 IM
PHAN, DUNG MY, 959 N EMPORIA S 2B,67214
267-5580
48 F 94101 75 FP
PHIPPS, JACK G, 315 N HILLSIDE,67214
686-3391
21 M 1902 53 FP
PICKENS, ANDREW T, WICHITA PSY PO BOX 8037,67208
684-0201
43 M 2834 69 D
PINSKER, JACOB A, 1035 N EMPORIA,67214
-
06 M 1902 35 IM
POLING, TERRY L, 6155 E HARRY,67218
685-2306
36 M 1902 62 FP
POLLACK, SIMON, 7523 PLAZA LANE,67206
-
M 36 OO
POLLANO DO, STEPHEN M, 1035 N EMPORIA #185,67214
265-5291
39 M 2878 68 PM
POLLOCK, ANTHONY G A, 1035 N EMPORIA STE 140,67214
265-1631
45 M 8305 71 ORS
POOLE, BERNARD T, 1035 N EMPORIA,67214
264-2805
37 M 53902 62 ORS
PORTER, GARRY L, 3243 E MURDOCK STE 400,67208
686-7351
35 M 1606 61 D
POWERS, K DEAN, 2703 E CENTRAL,67214
685-1277
23 M 1902 47 DBG
PRIETO, LUIS E, 959 N EMPORIA #203,67214
265-2513
39 M 26407 65 IM
PULLMAN, NORMAN K, 3007 E CENTRAL,67214
686-7369
21 M 3006 45 DS
PULLUM, RICHARD W, 2622 W CENTRAL,67203
945-9161
27 M 1875 57 R
PURINTON, LEW W, 1431 S BLUFFVIEW DR,67218
685-1301
23 M 1902 48 IM

PUTNAM, LYLE B, 1035 N EMPORIA,67214
262-5037
11 M 1902 36 FP
RAGHAVAN, PARULA P, 2404 GREENLEAF CT,67226
263-6131
47 F 49501 70 IM
RAGHAVAN, PRAKASH V, 1144 N ST FRANCIS,67214
267-0159
46 M 49501 69 CO
RANDALL, GEORGE R, 5205 E 21ST,67208
684-2838
43 M 2802 69 OTO
RANOLES, MICHAEL J, 959 N EMPORIA,67214
265-0789
48 M 1902 73 IM
RANSOM, KENNETH J, 905 N EMPORIA,67214
263-0296
49 M 1902 74 GS
RARDIN, MARY L, 737 S BROADVIEW,67218
52 F 1902 77 FP
RAUSA JR, FRANCISCO C, 1148 SOUTH HILLSIDE,67211
683-4658
42 M 74808 66 IM
RAVIPHANTHAKUK, KORCHI, 816 S EDGEMOOR,67218
49 M 1902
RAWCLIFFE JR, ROBERT A, 732 N TOPEKA,67214
267-1924
29 M 3501 55 ORS
RAZEK, ZACK A, 905 NORTH EMPORIA,67214
263-0296
46 M 60501 70 COTS
READER, G WHITNEY, PO BOX 2517,67201
263-5889
48 M 2101 70 IM
REALS, WILLIAM J, UKSM-WICHITA,67214
268-8224
20 M 3006 45 PATH
REAZIN, WALTER L, 1430 HOMESTEAD,67208
685-1381
30 M 1902 58 FP
REDDI, RAGHUNATH P, ST JOSEPH MEDICAL CTR,67218
685-1111
36 M 49521 64 RT
REED, A J, 1520 S CLIFTON,67218
685-2371
40 M 3901 65 EM
REED, D CRAMER, WESLEY MED CNTR/PROF DEV,67214
689-2080
15 M 2902 41 AOM
REED, DAVID O, 3333 E CENTRAL,67208
685-1291
43 M 1902 69 OR
REEVES, BRADFORD F, 3333 E CENTRAL,67208
685-1291
37 M 4812 62 R
REISMAN, MICHAEL ALAN, 3243 E MURDOCK SUITE 600,67208
683-5688
50 M 4804 75 OPH
RELIHAN, DONALD A, 3333 E CENTRAL,67208
684-5158
27 M 1902 54 OPH
REMPEL, JOHN H, 3333 E CENTRAL,67208
685-1812
38 M 3901 62 PS
RHOADS, JAMES P, 3244 E DOUGLAS,67208
689-9105
34 M 3520 60 IM
RHODEN, CURTIS H, 3243 E MURDOCK,67208
684-0252
33 M 1903 59 IM
RHODES, IVAN E, 3333 E CENTRAL,67208
685-1291
25 M 3901 49 R
RHODES, LOWELL M, 315 N HILLSIDE,67214
685-1461
25 M 1902 53 FP
RICHARDSON, STEWART F, 3243 E MURDOCK SUITE 400,67208
685-2321
28 M 300S 54 P
RIEDERER, ROBERT E, 1131 S CLIFTON,67218
685-4354
16 M 1902 42 FP
RIEGER, ERNEST H, 3243 E MURDOCK,67208
682-4591
29 M 1902 56 GS
RIEPE, ROGER E, WESLEY MED CENTER,67214
688-2810
46 M 1803 74 PATH
RIORDAN, HUGH D, 434 N OLIVER,67208
682-9241
32 M 5605 57 P

ROACH, NEIL E, UKSM - WICHITA,67214
258-8388
38 M 1902 67 CP
ROBERTS, DANIEL K, 3333 E CENTRAL SUITE 301,67208
688-3185
36 M 300S 61 OBG
ROBERTSON, JOSEPH K, 905 N EMPORIA,67214
263-0296
41 M 3901 66 GS
ROBINSON, G DONALD, 3333 E CENTRAL,67208
686-6659
28 M 1902 54 PD
ROBINSON, JOHN E, 2708 E CENTRAL,67214
686-7351
32 M 6201 50 P
ROBINSON, ROBERT H, 3244 E DOUGLAS,67208
689-9445
20 M 1902 53 ANES
ROBL, DAVID A, 10300 W MAPLE,67209
722-6892
48 M 1902 74 FP
RODRIGUEZ-RAMOS, ERNEST R, 1111 N ST FRANCIS,67214
265-2613
42 M 27501 67 COTS
RODRIGUEZTOCKER, LILIA, 1111 N ST FRANCIS,67214
265-2613
21 F 27501 49 IM
ROMALIS, BRIAN E, 3429 E DOUGLAS,67218
682-5069
39 M 6201 63 P
ROOS, MAUREEN, 1122 N TOPEKA,67214
265-2876
53 F 1902 75 FP
ROSE, SHELBY O, 3333 E CENTRAL,67208
681-2741
40 M 2012 68 PATH
ROSEN, DAVID, 1035 N EMPORIA STE 265,67214
265-3226
48 M 1902 74 PD
ROSENBERG, THOMAS F, 2627 E CENTRAL,67214
684-0501
41 M 1642 68 A
ROSS, DENNIS LEE, 103S N EMPORIA 105,67214
263-7285
47 M 300S 73 NEP
RUSSELL, PHILIP W, 3244 E DOUGLAS,67208
689-9351
22 M 1902 44 IM
SABIN JR, GEORGE M, 707 N MAIN,67203
265-6601
12 M 5002 39 AOM
SADIQ, SULEMAN, 1144 N ST FRANCIS,67214
267-0159
40 M 70401 63 TS
SAEED, MOHAMMAD, 112S S CLIFTON,67218
685-2371
42 M 70404 66 IM
SANDERS, GLORIA DEANNA, OSTEOPATHIC HOSPITAL,67212
945-9161
50 F 1902 75 PATH
SANTOSCOY, GILBERT S, 3244 E DOUGLAS,67208
689-9124
38 M 4812 62 GS
SCANLAN, TIMOTHY M, 1122 N TOPEKA,67214
265-2876
46 M 2604 71 FP
SCHILTZ, FRANCES, 115 S RUTAN #108,67218
93 F 6701 23 OD
SCHLACHTER, ERNEST R, 406 E CENTRAL,67202
265-0705
24 M 1902 52 FP
SCHLICHER, JOHN E, 3244 E DOUGLAS,67208
689-9346
40 M 1903 66 O
SCHLUETER, JOHN J, 3333 E CENTRAL,67208
685-9289
31 M 3841 56 R
SCHNELLE, JOACHIM, 4145 EAST KELLOGG,67218
682-0621
44 M 40933 70 FP
SCHOPF, CLIFTON C, FAMILY PRACTICE CLINIC,67214
262-3765
29 M 1902 57 FP
SCHWARTZ, V DEAN, 400 N WOODLAWN #4,67208
684-3881
24 M 1902 48 FP
SCOTT, VINCENT L, 72 MISSION RD,67207
03 M 3806 29 OD
SCOTT, WILLIAM H, 1431 S BLUFFVIEW STE 111,67218
685-1111
41 M 4901 65 CD

SEN SARMA, PRONAB K, 1144 N ST FRANCIS,67214
267-0159
45 M 49518 71 CD
SHAFER, PRESTON J, 3333 E CENTRAL,67208
682-6511
20 M 3005 46 OBG
SHAH, MUKHTAR H, 841 S HILLSIDE,67211
686-2994
40 M 70404 63 P
SHAW, RICHARD C, 825 N EMPORIA,67214
262-3495
35 M 1902 61 PS
SHELLITO, JOHN G, 3244 E DOUGLAS,67208
689-9124
18 M 1506 43 TS
SHIELD, CHARLES, 905 N EMPORIA,67214
263-0295
46 M 2802 72 GS
SHRADER, DOYLE A, 3333 E CENTRAL,67208
682-4851
16 M 1902 41 EENT
SHURTZ, GLEN L, 3333 E CENTRAL SUITE 214,67208
685-1291
40 M 4802 78 R
SIEGEL, ALBERT R, 3600 E HARRY,67218
685-1111
22 M 1642 47 PM
SIFFORD, R LAWRENCE, 959 N EMPORIA,67214
265-0561
25 M 1803 52 IM
SKIBBA, RICHARD M, 3244 E DOUGLAS,67208
689-9477
43 M 5606 70 GE
SLUTSKY, LAWRENCE JOEL, ST FRANCIS HOSPITAL,67214
268-5922
46 M 3501 72 DR
SMITH, ALVIN L, 929 N ST FRANCIS,67214
262-6211
28 M 5606 57 PATH
SMITH, TIMOTHY WM, 1035 N EMPORIA #185,67214
265-5291
49 M 1902 74 IM
SMITH JR, WILLARD J, 851 N HILLSIDE,67214
685-1371
32 M 1511 57 U
SNYDER, GREGG M, 902 N HILLSIDE,67214
685-2377
27 M 1803 54 NS
SOLOMON, HERMAN, 835 N HILLSIDE,67214
685-4395
37 M 2701 62 D
SOLTZ, ROBERT A, 3244 EAST DOUGLAS,67208
689-9381
47 M 2803 74 PD
SOMERS, MARVIN M, ST FRANCIS HOSP,67214
252-6211
23 M 1902 48 R
SPANN, RICHARD W, 3243 E MURDOCK,67208
684-0252
40 M 1902 65 PUD
STANLEY, KENNETH E, 959 N EMPORIA,67214
267-0256
31 M 1902 56 U
STARK, JAMES R, 3244 E DOUGLAS,67208
689-9422
20 M 1902 44 R
STECKLEY, RICHARD ALLEN, 1035 EMPORIA SUITE 130,67214
263-3271
49 M 2105 74 IM
STEIN, PAUL S, 3243 E MURDOCK,67208
685-2377
40 M 3305 66 NS
STEMBRIDGE, TRAVIS W, 3333 E CENTRAL SUITE 301,67208
685-7234
47 M 4802 76 OBG
STEPHANZ JR, GERALD B, 834 CARTER,67203
265-5554
57 M 1902
STOLZ, ELMER G, 2349 MC LEAN BLVD NW,67204
265-6287
17 M 3006 45 FP
STREET, DAVID E, 905 N EMPORIA,67214
263-0296
35 M 2101 61 GS
STREIT, JEROME G, 1131 S CLIFTON,67218
685-4354
48 M 1902 77 FP
STRICKLAND, MAURICE VAN, 3244 E DOUGLAS,67208
689-9156
51 M 4804 74 A
STRYKER, TERRY MARGARET, 3243 E MURDOCK - STE 300,67208
685-8231
47 F 1002 75 FP
SUERO, JESUS T, 1152 SOUTH CLIFTON,67218
681-1671
33 M 74802 57 PUD
SULLIVAN, CORNELIUS J P, 1431 BLUFFVIEW DR,67218
685-4851
19 M 3509 43 OBG
SULLIVAN, LEONARD L, 3244 E DOUGLAS,67208
689-9454
35 M 1902 61 PD
SVOBODA, LOIS V, 3243 E MURDOCK STE 300,67208
685-8231
39 F 1602 66 FP
SVOBODA, WILLIAM B, 3243 MURDOCK SUITE 601,67208
685-2377
36 M 1602 63 PDN
SWISHER, WILLIAM C, 1425 WILLOW LANE,67208
682-1534
21 M 1902 47 OBG
TATPATI, DANIEL A, 1144 N ST FRANCIS,67214
267-0159
44 M 49535 67 TS
TATPATI, OLGA ADELINA, U K S M - WICHITA,67214
268-5992
44 F 49535 67 PD
TAYLOR, RICHARD J, 929 N ST FRANCIS,67214
262-1952
21 M 3006 49 PATH
TAYLOR, STEVEN L, USAF HOSP,67210
681-5866
45 M 1902 77 GP
THELEN, J CHRISTINE, 1738 N ROOSEVELT,67208
-
13 F 5104 37 OO
THOMPSON, DANIEL M, PO BOX 4044,67204
838-3381
19 M 1902 50 FP
THOMPSON, SCOTT W, 1632 W 22ND #407,67204
838-0994
51 M 64914 78 FP
TIHEN, EDWARD N, 3244 E DOUGLAS,67208
689-9481
24 M 1606 48 IM
TIHEN, HENRY N, 1227 N RIVER BLVD,67203
-
96 M 1601 19 OO
TILLER, GEORGE R, 5101 E KELLOGG,67218
684-5255
41 M 1902 67 AM
TILTON, FRANK M, 3244 E DOUGLAS,67208
689-9137
33 M 2002 59 N
TINKER, ROBERT C, 1126 S CLIFTON,67218
682-7578
31 M 2834 57 ORS
TINTEROW, MAURICE M, 3333 E CENTRAL,67208
685-4389
17 M 4802 41 ANES
TIPPIN JR, ERNEST E, 959 N EMPORIA,67214
265-5256
24 M 1902 50 OTO
TOCKER, ALFRED M, 1111 N ST FRANICS,67214
265-2613
15 M 4802 40 CDTS
TONN, GERHART R, 855 N HILLSIDE,67214
685-1381
16 M 1902 44 FP
TOSH, FRED E, 1900 E NINTH,67214
258-8391
30 M 4706 54 PH
TRACY, TERRY A, 3333 E CENTRAL,67208
686-7301
35 M 2803 61 OBG
TRETBAR, HARVEY A, 3243 E MURDOCK #500,67208
684-0251
25 M 1902 52 IM
TREWEEKE, MICHAEL W, 2916 EAST CENTRAL,67214
684-5234
46 M 1902 72 IM
TRUDEAU, DAVID L, ALCOHOL TREATMENT UNIT,67218
685-1111
40 M 2504 66 ADT
TRUJILLO, ANTERO A, 3600 E HARRY,67218
685-1111
35 M 73701 61 ANES
UHLIG, PAUL J, 3244 E DOUGLAS,67208
689-9191
28 M 1902 57 PD
ULRICH, BRIAN K, 1035 N EMPORIA,67214
265-3226
43 M 1902 74 IM
VAN LEEUWEN, GERALD, UKSM-WICHITA,67214
268-8221
29 M 1803 54 NEO

VIN ZANT, LARRY E, 1431 BLUFFVIEW STE 109,67218
682-6532
10 M 1902 40 GS
VINE, DONALD LEE, 3244 E DOUGLAS,67208
689-9240
39 M 511 66 CD
VINZANT, WHITNEY L, 1431 SOUTH BLUFFVIEW #109,67218
686-1991
45 M 1902 71 GS
VORHEES, VICTOR J, 3243 E MURDOCK,67208
685-8231
36 M 1902 68 FP
VOTH, DOUGLAS W, UKSM-WICHITA DEPT OF IM,67214
268-8378
34 M 1902 59 IM
WAOUD, ABOUL, 1543 S HILLSIDE,67211
682-6814
35 M 70409 60 P
WALTERS, ROBERT MERRILL, 3244 E DOUGLAS,67208
789-9495
41 M 2802 74 ORS
WARD, L G, 1201 N RIVER BLVD,67203
263-1574
54 M 1902 79 ANES
WARREN, LLOYD P, 5205 E 21ST,67208
683-7223
11 M 1902 36 OPH
WARREN, WIRT A, 352 N BROADWAY,67202
267-0520
09 M 2802 33 GER
WARREN JR, JOHN W, 3244 E DOUGLAS,67208
689-9239
15 M 2501 39 U
WEAVER, J ROBERT, 959 N EMPORIA,67214
265-5731
21 M 1902 48 FP
WEAVER, JACK O, 959 N EMPORIA,67214
265-7241
16 M 2802 42 OPH
WEBER JR, HUGO P, 1035 N EMPORIA,67214
263-7285
40 M 702 66 IM
WEBSTER, BOBBY W, 550 N LORRAINE,67214
685-0559
48 M 4802 74 OBG
WEIPPERT, EDWARD J, 10300 W MAPLE,67209
722-6892
44 M 1902 70 FP
WELCH, ANNA L, 130 N SEDGWICK,67203
-
48 F 1720 81 FP
WELCH, LAUREN K, 3243 E MURDOCK SUITE 601,67208
685-2377
35 M 1902 61 N
WELCH, MARTIN H, UKSM-WICHITA,67214
268-8378
36 M 5605 61 IM
WELLS, MAX MICHAEL, 550 N HILLSIDE,67214
688-2820
50 M 1902 76 PATH
WELLSHEAR, CHARLES C, WICHITA PSY CENTER,67208
684-0201
30 M 4706 58 P
WENINGER, JOHN H, 1148 S HILLSIDE,67211
682-6523
32 M 3005 62 FP
WEST, WILLIAM T, 3244 E DOUGLAS,67208
689-9234
24 M 1902 49 OBG
WHALLON, JACOB T, 202 N ROCK RD APT 120,67206
-
14 M 1720 41 OO
WHEELER, NICKY RAY, 3007 E CENTRAL,67214
686-7369
48 M 1902 74 PS
WHEELER, PINCKNEY R, 2208 W 13TH,67203
943-2118
18 M 3901 56 FP
WHITAKER, JAMES A, 3243 E MURDOCK,67208
684-0251
44 M 1902 72 IM
WHITE, CHARLES M, 3244 E DOUGLAS,67208
689-9422
15 M 3005 41 R
WILCOX, ROBERT N, 841 N BROADWAY,67214
263-6131
27 M 1902 54 R
WILDER, LOWELL W, 3333 E CENTRAL,67208
684-5158
35 M 4109 62 OPH
WILKINSON, LARRY K, 1520 S CLIFTON,67218
685-2371
46 M 1902 74 FP
WILLIAMS, CHARLES L, 3244 E DOUGLAS,67208
689-9269
15 M 2834 43 IM
WILLIAMS, HOWARD V, 1439 GOEBEL CIRCLE,67207
268-8251
18 M 1205 43 P
WINCHELL, H H FORSYTH, P O BOX 11344,67211
265-1684
34 M 3545 58 R
WISDOM, JAY K, 1148 S HILLSIDE,67211
684-0271
12 M 1902 42 FP
WISNER JR, HARRY J, 3244 E DOUGLAS,67208
689-9169
17 M 3005 43 IM
WITTMANN, ALBERT F, 2323 N WOODLAWN,67220
-
10 M 2834 38 OO
WOLFE, FREDERICK, 6611 E CENTRAL,67206
686-1152
36 M 3508 66 RHU
WOOD, GARY B, 3243 E MURDOCK #404,67208
684-2131
21 M 2802 45 IM
WOOD, ROBERT D, 2337 RUTLAND CT,67226
486-2127
26 M 1902 53 FP
WOODHOUSE, CHARLES L, 959 N EMPORIA,67214
265-8821
10 M 1902 34 ENT
WORSING JR, ROBERT A, 3244 E DOUGLAS,67208
689-9175
47 M 2604 72 ORS
WRAY, ALEXANDER J, 120 E 21ST ST,67214
838-4912
19 M 1902 49 FP
WU, JIN-TZE, 3333 E CENTRAL SUITE 214,67208
688-2920
41 M 38502 67 TR
YOCKEY, CHARLES C, 3243 E MURDOCK S 500,67208
684-2051
45 M 1902 72 IM
YOON, CHANG SUP, 3333 E CENTRAL SUITE 602,67208
688-2640
46 M 58303 72 ANES
YOUNG, DOUGLAS L, 3244 E DOUGLAS,67208
689-9213
42 M 1902 71 IM
YOUNG, ROBERT, 3243 E MURDOCK,67208
688-5211
48 M 1902 78 OBG
YOUNGBERG, DEAN I, 2916 EAST CENTRAL,67214
684-5243
46 M 1902 72 IM
ZARNOW, HILARY, 1624 GATEWOOD,67206
263-3219
45 M 1611 69 R
ZATZKIN, JAY B, 1035 N EMPORIA,67214
252-4467
46 M 2002 74 IM
ZEPICK, LYLE F, 933 N TOPEKA,67214
263-5889
50 M 6001 71 CD
ZIMMERMAN, KENNETH D, 934 CRESTLINE,67212
687-3925
29 M 3901 65 OM
ZONGKER, PHILIP E, 3244 E DOUGLAS,67208
689-9422
43 M 1902 70 R

WILSON—913
(Central Kansas Society)

OLABAL, FRANK A, PO BOX 160,67490
658-2400
07 M 1902 45 FP

WINCHESTER—913
(Jefferson County Society)

HUSTON, FRANCIS W, ,66097
-
06 M 1501 34 FP

WINFIELD—316
(Cowley County Society)

CARRO, F AURELIO, PO BOX 544,67156
221-3200
23 M 27501 49 IM

OAEHNKE, SIGURD S, PO BOX 544,67156
221-3200
26 M 3005 60 FP
FOWLER, DENNIS L, PO BOX 544,67156
221-2300
48 M 1902 73 ES
HUTCHINSON, DIRK T, PO BOX 544,67156
221-3200
43 M 3901 74 IM
KAUFMAN, LELAND R, 221 WEST 8TH,67156
221-3350
33 M 1902 61 FP
KAUL, ANAND N, PO BOX 544,67156
221-3200
39 M 49530 61 IM
LAWRY, JAMES VORIS, 1017 E EIGHT,67156
221-3200
40 M 502 77 GP
MAC KILLOP JR, DANIEL, 4 FLEETWOOD DR,67156
-
11 M 2407 38 OO
MILLER, FRANKLIN R, BOX 544,67156
-
02 M 2401 27 IM
NEMMERS, DAVID J, PO BOX 544,67156
221-3200
34 M 1803 59 IM
SAMUEL, CHANOY C, 1211 E 5TH,67156
221-6100
35 M 49527 59 GS
SAMUEL, SHANTHI, 5 FLEETWOOD DR,67156
221-2300
42 F 49527 67 PATH
SHAH, ASHOK H, PO BOX 544,67156
221-3200
41 M 4954B 66 OBG
SNYDER, C JOHN, 1317 WHEAT RD,67156
221-3200
38 M 2401 64 GS
SNYDER, HOWARD E, 1317 WHEAT ROAD,67156
221-3200
03 M 4102 27 GS
WELLS, ALVIN Y, 1707 E 10TH,67156
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97 M 1902 37 OO
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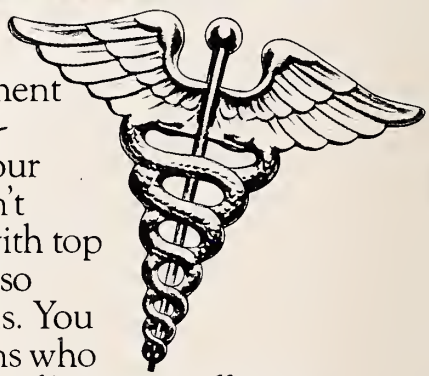
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The Physician's Sleep Glossary

Some common sleep laboratory terms

poly•som•no•graph. An instrument which simultaneously records by electrodes physiological variables during sleep—for example, brain activity (EEG), eye movements (EOG), muscle tonus (EMG) and other electrophysiological variables. These readings indicate precisely when patients fall asleep, how many wake periods they experience, the quality of sleep and the duration of sleep.

sleep la•ten•cy. The period of time measured from "lights out," or bedtime, to the commencement or onset of sleep.

wake time af•ter sleep on•set. Intervals of time spent awake between onset of sleep and the end of the sleep period. The polysomnograph registers the length and frequency of the intervals.

to•tal sleep time. The amount of time actually spent in sleeping. This is estimated by subtracting wake times from the period encompassed by the onset and the termination of sleep.[1]

REM/NREM. 1. REM, or rapid eye movement, sleep is "active"—characterized by increased metabolic rates, elevated temperature and arousal-type EEG patterns. 2. NREM, or non-rapid eye movement, sleep represents "quiet" sleep stages. There are four distinct stages of NREM sleep.[2]

re•bound in•som•nia. A statistically significant worsening of sleep compared to baseline on the nights immediately following discontinuation of sleep medication.[3]

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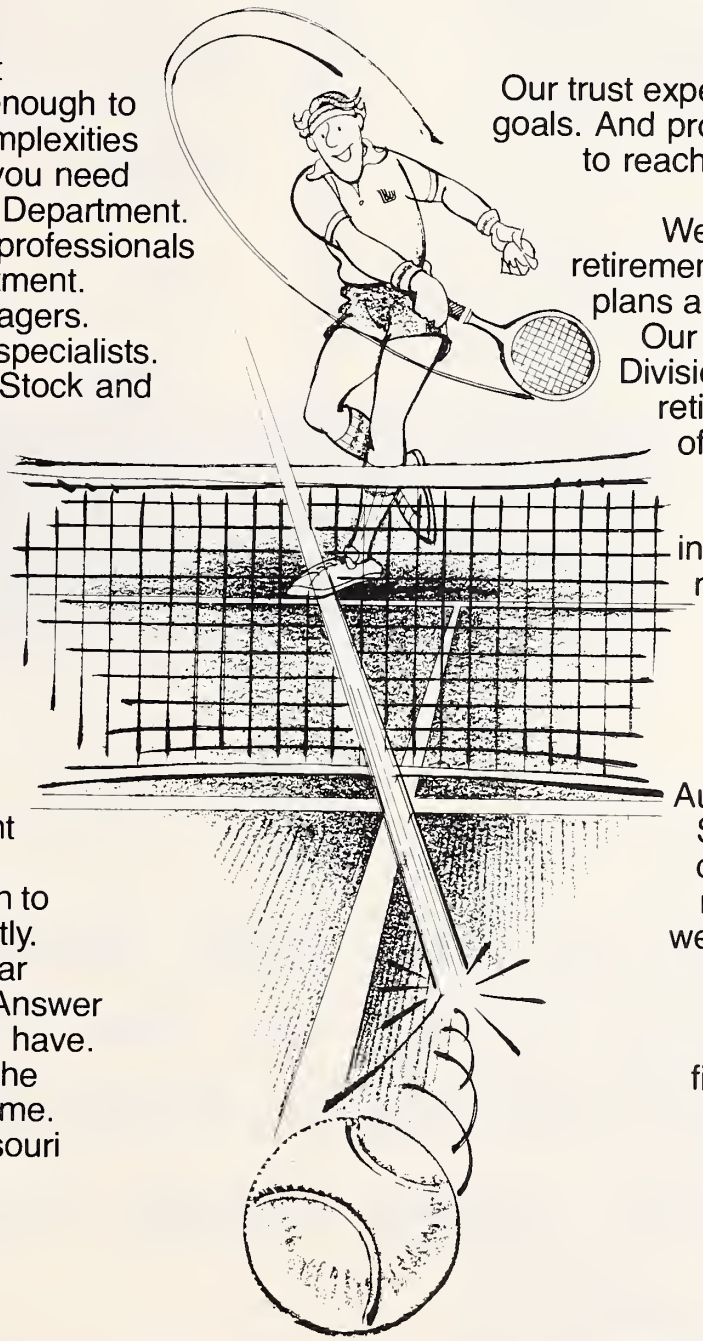
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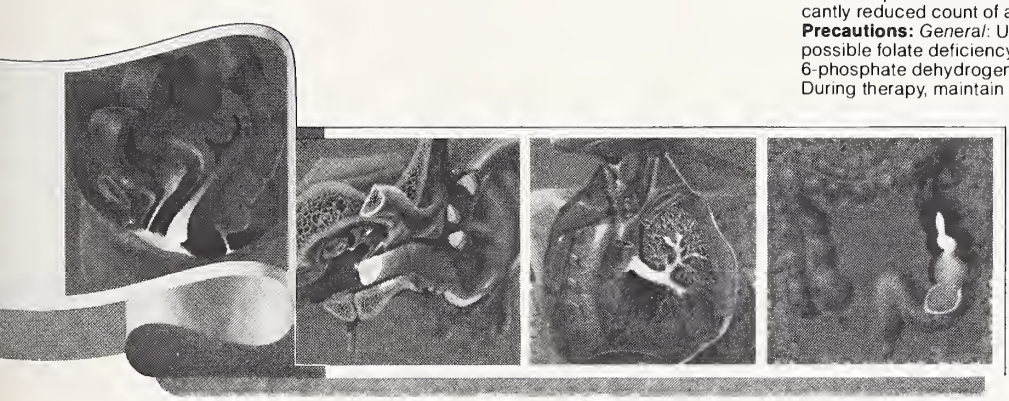
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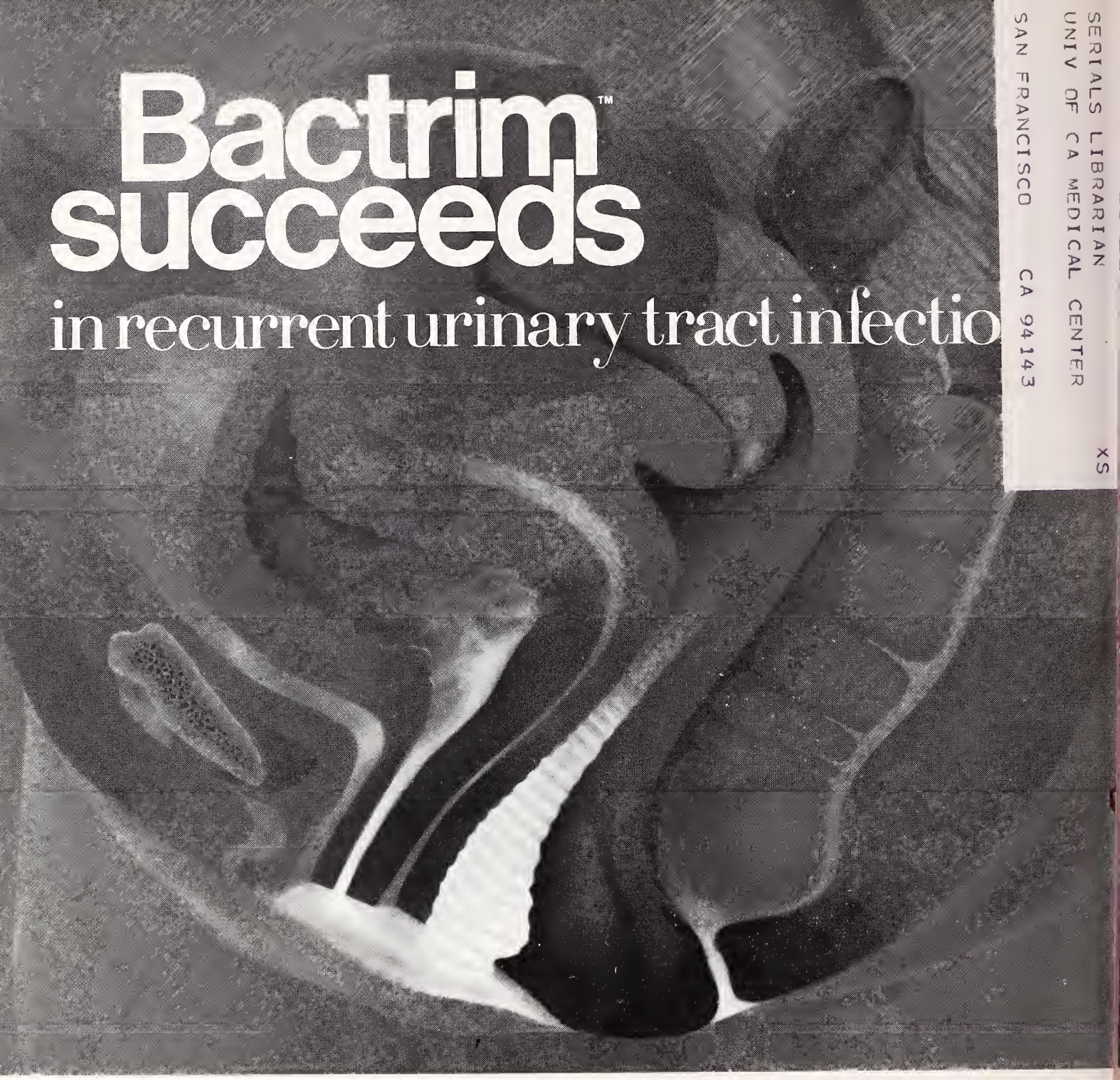
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For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.
For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.
Also for the treatment of documented *Pneumocystis carinii* pneumonitis.
Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.
Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.
Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function. Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.
Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.
Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.
Dosage: Not recommended for infants less than two months of age.
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:
Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.
Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.
For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:
Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.
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1. Rubin RH, Swartz MN: *N Engl J Med 303*:426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

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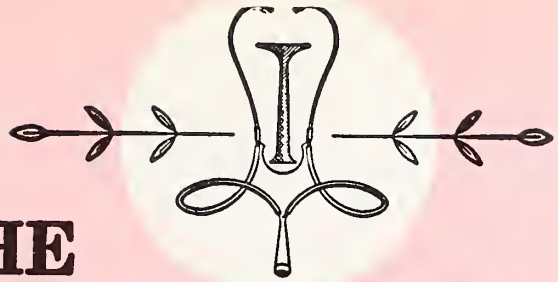
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AMA House of Delegates

Summary of Actions Taken at the 1982 Annual Meeting, Chicago

The 1982 Annual Meeting of the American Medical Association was held on June 13-17 in Chicago. The Kansas Medical Society was officially represented by its full complement of delegates (the undersigned), as well as Kermit G. Wedel, M.D., President, Steve Carter and Jerry Slaughter, Staff. Also present from Kansas was William J. Reals, M.D., Wichita, representing the College of American Pathologists, and Franklin G. Bichlmeier, M.D. and George E. Burket, Jr., M.D.

The 236 Delegates present represented state medical organizations, 61 specialty societies, and eight sections and service groups.

Kansas was successful in promoting the re-election of William J. Reals, M.D., Wichita, for a seat on the AMA Council of Medical Education. This council is concerned with undergraduate, graduate, and postgraduate education in the United States. It is comprised of 12 physicians and staff members, and meets regularly to discuss medical education matters and to advise the AMA on major policy issues.

Some of the major issues considered by the House were the following:

National Health Policy

The House approved AMA development of a National Health Policy over a two-year period. A variety of professional, business, labor, and insurance organizations will participate. The purpose is to establish the private sector's national agenda for dealing with health issues. A set of basic principles and a Health Policy Plan will be reviewed by AMA Councils, the Board, and the AMA House.

Federal Trade Commission

The House adopted two reports and three resolutions calling for continued strong support for federal legislation pertaining to FTC jurisdiction over the learned professions:

- S.2499 would reauthorize the FTC; amend the FTC act to specify that there is no FTC jurisdiction over the professions; prohibit FTC pre-emption of state law; and make procedural reforms in FTC proceedings.
- S.1984 provides clearly that the FTC does not have jurisdiction over the professions and cannot pre-empt state laws relating to the professions. These provisions have been offered as an amendment to S.2499.
- H.R.3722 (Luken-Lee Bill) would place a moratorium on FTC activities regarding state-regulated professions and professional associations.

Hospital Medical Staff Representation

The Board recommended a Section on Medical Staffs in the House, concluding that organized medical staffs need to be strengthened. The House authorized a Study Committee to submit alternative mechanisms to the Interim Meeting.

AMA Dues — 1983

The House approved a Board of Trustees proposal to increase regular dues by $30 in 1983. Other dues categories will be increased proportionately except for medical students. Dues for resident physicians will be increased by $10.

In recommending the dues increase, the Reference Committee noted the economic condition of the country, but said that the work of the Association is important and can only continue successfully from a firm financial position. They reported that there was no decrease in membership last year when the dues were raised in the first of the planned increments.

Young Physicians in Organized Medicine

The House received a progress report on young physician participation in organized medicine. This report recommended modification of some membership procedures that exist in the federation. These include: That the AMA strongly urge all county and state societies to review their bylaws and membership procedures and remove all unnecessary barriers to membership as soon as possible; that the AMA endorse and encourage the universal utilization of the Uniform Application and Billing Forms recently developed by AAMSE; and that the AMA encourage all county and state societies to implement a simple "transfer of memberships" procedure that will facilitate uninterrupted memberships in organized medicine for physicians who relocate at any time during their careers.

(Continued on page 455)

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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.
Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation. The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
How Supplied: For oral administration, Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100* and 500;* Prescription Paks of 50, available in trays of 10.* Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25,† and in boxes containing 10 strips of 10.†
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Vox Dox

Vox Dox Editor:

I cannot resist commenting on the article "Mandatory CME" (*J. Kans. Med. Soc.* 83:295, 1982). I feel it is vitally important for our Society to view continuing medical education (CME) in its proper perspective. I believe the study reported informs us about the participation of Kansas physicians in CME and what sort of courses appeal to them. I feel, as is stated, that the only real test of the effectiveness of CME is improved patient care.

It is important to realize the amount of time and money devoted to CME. One need only reflect on the numbers of colorful brochures describing exotic locales that cross one's desk on a daily basis to realize that CME is a multimillon dollar business. We can take pride in Kansas that our state medical educational facility has been one of the leaders in providing postgraduate education to Kansas physicians for 40 years, not just four. Circuit courses and meetings have followed the format Kansas physicians like — that of the traditional lecture. Those courses now have varied and expensive competition. The question is, how much good does it all do?

Kansas was one of the early riders of the bandwagon requiring CME. The result has been a growth in geometric proportion of CME opportunities. Our local hospitals have hired educational gurus and are building extravagant temples in which to worship CME at home. But is it good because the AMA says so and the KMS requires it? There is no evidence to prove that CME reduces morbidity or mortality or hospital stay. All of us do it because it feels good. We get to socialize with our colleagues, have coffee, and take notes. Our training taught us that taking notes equals learning. But do we change our professional behavior? Studies would indicate that we do not.

It is apparent that we need to re-evaluate the traditional lecture format CME as a requirement for licensure. For some it may be a good learning experience, so the lecture format should be retained. Perhaps we need more emphasis on other methods of learning such as the mini-residency or the resources of the fascinating computer.

Lastly, how do we measure whether or not a physician is keeping up? The Academy of Family Practice and now other specialties require recertification. Does passing an examination insure competence of a physician any more than attending a course? Probably not, as only a segment of informa-

(Continued on page 458)

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CARDIAC FAILURE: In congestive heart failure, inhibition with beta-blockade carries the potential hazard of further depressing myocardial contractility and precipitating cardiac failure. In patients already receiving digitalis, propranolol may reduce the positive inotropic action of digitalis and may have an additive depressant effect on AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, in rare instances, cardiac failure has developed during propranolol therapy. At the first sign of impending cardiac failure, patients should be fully digitalized and/or given a diuretic, and observed closely a) if cardiac failure continues, despite adequate digitalization and diuretic therapy, propranolol should be immediately withdrawn; b) if tachyarrhythmia is being controlled, patients should be maintained on combined therapy and closely followed until threat of cardiac failure is over.

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuation of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when INDERAL is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease, who are given propranolol for other indications.

IN PATIENTS WITH THYROTOXICOSIS, possible deleterious effects from long term use have not been adequately appraised. Give special consideration to propranolol's potential for aggravating congestive heart failure. Propranolol may mask the clinical signs of developing or continuing hyperthyroidism or complications and give a false impression of improvement. Propranolol should be withdrawn slowly, since abrupt withdrawal may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

IN PATIENTS UNDERGOING MAJOR SURGERY, beta-blockade impairs the ability of the heart to respond to reflex stimuli. Except in pheochromocytoma, propranolol should be withdrawn 48 hours prior to surgery. In case of emergency surgery, the effects of propranolol can be reversed by administration of beta-receptor agonists such as isoproterenol or levarterenol, but such patients may be subject to protracted severe hypotension. Difficulty in restarting and maintaining the heart beat has been reported.

IN PATIENTS PRONE TO NONALLERGIC BRONCHOSPASM (*e.g.*, CHRONIC BRONCHITIS, EMPHYSEMA), administer with caution, since propranolol may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta-receptors.

DIABETICS AND PATIENTS SUBJECT TO HYPOGLYCEMIA. Propranolol may prevent the appearance of premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia, especially in patients with labile diabetes. A precipitous elevation of blood pressure may accompany hypoglycemic attacks.

USE IN PREGNANCY: Safe use in human pregnancy not established. Embryotoxic effects have been seen in animals at doses about 10 times the maximum recommended human dose.

PRECAUTIONS

Patients receiving catecholamine depleting drugs such as reserpine should be closely observed if propranolol is administered, since it may occasionally produce hypotension and/or marked bradycardia resulting in vertigo, syncopal attacks, or orthostatic hypotension.

Observe laboratory parameters at regular intervals. Use with caution in patients with impaired renal or hepatic function.

ADVERSE REACTIONS

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; arterial insufficiency, usually of the Raynaud type; thrombocytopenic purpura. *Central Nervous System:* lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. *Gastrointestinal:* nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis. *Allergic:* pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress. *Respiratory:* bronchospasm. *Hematologic:* agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura. *Miscellaneous:* reversible alopecia. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta-blocker (practolol) have not been conclusively associated with propranolol. *Clinical Laboratory Test Findings:* Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

HOW SUPPLIED

TABLETS

—Each hexagonal-shaped, orange, scored tablet is embossed with an "I" and imprinted with "INDERAL 10," contains 10 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0421-81) and 1,000 (NDC 0046-0421-91). Also in unit dose package of 100 (NDC 0046-0421-99).

—Each hexagonal-shaped, blue, scored tablet is embossed with an "I" and imprinted with "INDERAL 20," contains 20 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0422-81) and 1,000 (NDC 0046-0422-91). Also in unit dose package of 100 (NDC 0046-0422-99).

—Each hexagonal-shaped, green, scored tablet is embossed with an "I" and imprinted with "INDERAL 40," contains 40 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0424-81) and 1,000 (NDC 0046-0424-91). Also in unit dose package of 100 (NDC 0046-0424-99).

—Each hexagonal-shaped, yellow, scored tablet is embossed with an "I" and imprinted with "INDERAL 80," contains 80 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0428-81) and 1,000 (NDC 0046-0428-91). Also in unit dose package of 100 (NDC 0046-0428-99).

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Store at room temperature (approximately 25° C).

INJECTABLE

—Each ml contains 1 mg of propranolol hydrochloride in Water for Injection. The pH is adjusted with citric acid. Supplied as 1 ml ampuls in boxes of 10 (NDC 0046-3265-10).

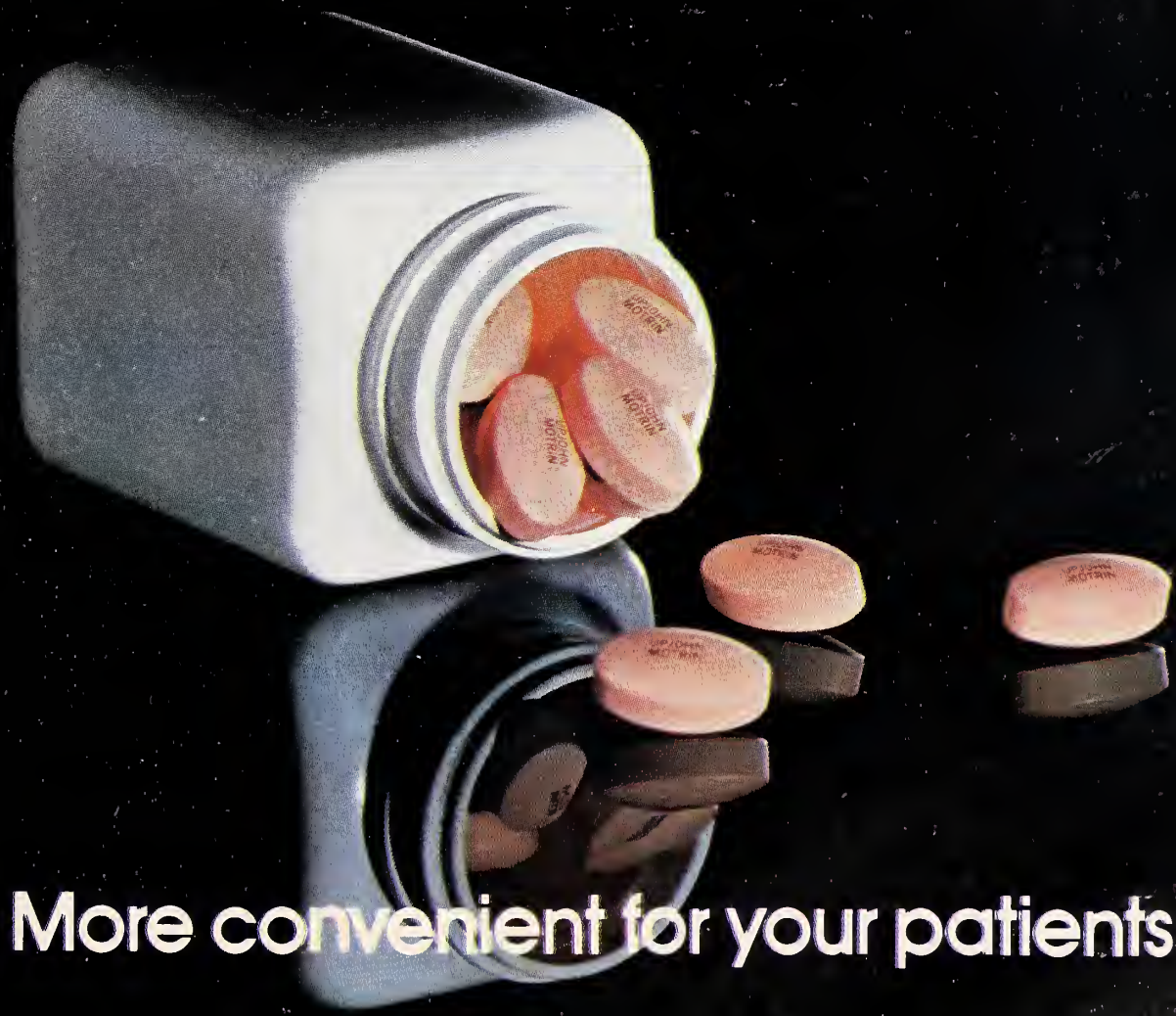
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Reference: 1. Freis, E.D.: Hypertension (Suppl. II) *3*:230 (Nov.-Dec.) 1981.

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The Physician's Sleep Glossary

Some common sleep laboratory terms

poly•som•no•graph. An instrument which simultaneously records by electrodes physiological variables during sleep—for example, brain activity (EEG), eye movements (EOG), muscle tonus (EMG) and other electrophysiological variables. These readings indicate precisely when patients fall asleep, how many wake periods they experience, the quality of sleep and the duration of sleep.

sleep la•ten•cy. The period of time measured from "lights out," or bedtime, to the commencement or onset of sleep.

wake time af•ter sleep on•set. Intervals of time spent awake between onset of sleep and the end of the sleep period. The polysomnograph registers the length and frequency of the intervals.

to•tal sleep time. The amount of time actually spent in sleeping. This is estimated by subtracting wake times from the period encompassed by the onset and the termination of sleep.[1]

REM/NREM. 1. REM, or rapid eye movement, sleep is "active"—characterized by increased metabolic rates, elevated temperature and arousal-type EEG patterns. 2. NREM, or non-rapid eye movement, sleep represents "quiet" sleep stages. There are four distinct stages of NREM sleep.[2]

re•bound in•som•nia. A statistically significant worsening of sleep compared to baseline on the nights immediately following discontinuation of sleep medication.[3]

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

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At the expiration date of your policy year, an audit is made by the insurance company to determine the actual payroll amounts, or other exposures during the year. Following this audit, an adjustment may be made which will require additional premium or a return or credit will be ordered. The following are five tips to assist you in preparing for an audit. The following sources will help the auditor:

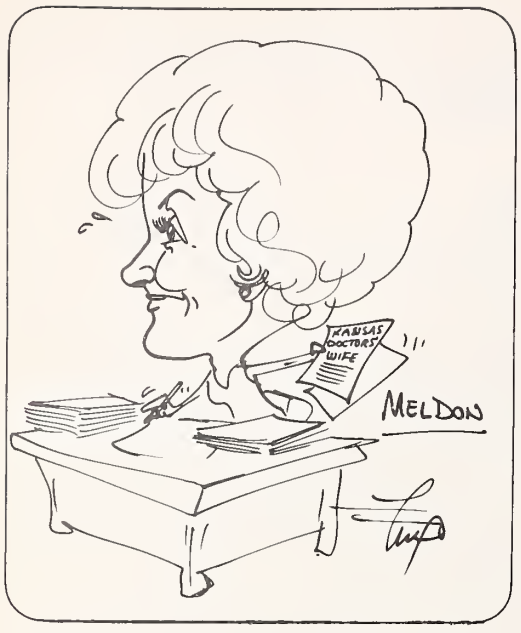
1. Payroll journal providing monthly totals and division of payroll by type of work performed.
2. Individual earning records indicating the type of work performed. Gross payroll should be totaled by the quarter.
3. Separate record of overtime shown by employee and totaled by class of work for the policy term involved. (Premium for Workers' Compensation is based on straight time pay for all hours worked and does not include ½ extra pay for overtime.) (Not applicable in Delaware, Pennsylvania, and Utah.)
4. Certificates of Workers' Compensation Insurance for all insured sub-contractors.
5. Social Security (Form 941) and State Unemployment Compensation quarterly returns.

Our auditors are instructed to inform you of the date they intend to call on you or to arrange in advance for a convenient time. To assure accurate assignment of your payroll to the proper classes, it is wise for you to arrange to have someone in your organization familiar with employee job assignments available to work with our auditor during the course of the audit.

If your records are kept by an outside accounting firm, make certain the accountants are aware of the impending visit by the auditor so they will have your records available when needed. In the event the accountant is not well informed regarding the duties of various employees, you may wish to brief him in advance of the auditor's visit.

In the audit of your payroll for final billing purposes, you need to determine that the payroll of individual employees is assigned to the appropriate rating classification. This assures that you will be paying the correct premium.

Annual premiums in excess of a specified amount qualify for a discount which varies by state and also by the amount of premium needed to be eligible for the discount. Contact your sales representative if you have any questions about discounts or classifications.



AUXILIARY NEWS

Forty-five members and guests attended Summer Conference in Ottawa June 22-23. Mary Lynn Gollier (Bud) served as Conference Chairman. Members were housed in dormitory rooms on the campus of Ottawa University. From the comments, we believe it was a productive and interesting time together.

Two committee meetings were held the first morning, followed by lunch served by the Franklin County Auxiliary members. A Board of Directors' meeting was held in the afternoon to conduct necessary business, followed by an address by our national representative, Mary Kay McPhee (William), North Central Regional Vice President, Kansas City, Missouri. Our own Jean Cavanaugh (Clair), North Central Regional Director, also spoke. State chairmen and delegates to convention gave reports.

A social hour in the Laury home preceded a bus trip to Vassar Junction for dinner and theatre. Following dinner on the Rogers train, we attended opening night of "Da" at the barn playhouse. The next morning, members of the Franklin County Auxiliary prepared a delicious breakfast at the home of Nelle Gollier (Bob).

The Conference reconvened in the Student Union at 9 AM with a Round Table Discussion by five past state presidents. Each recalled the emphasis of the Auxiliary during her year as president. There was also reminiscing about some of the lighter moments.

After coffee break, Kermit Wedel, M.D., President of KMS, spoke to us about some of the concerns of the Society. KMS is fortunate to have such an articulate speaker represent you. Val Braun, our liaison from KMS, gave an excellent talk with pertinent information. Steve Carter impressed our group with his dynamic personality. We were delighted that Gary Caruthers also was present. Roger Warren, M.D., gave an enthusiastic talk about KaMPAC. Our national representative was impressed that so many officials from the Kansas Medical Society came to speak at an auxiliary conference.

Luncheon followed with music by the "Prairie Grass Pickers" — Diane and Dr. Alan Sanders of Lawrence — who strummed their banjoes to bluegrass tunes. We were all a little amazed that they were so good!

We had good attendance of county presidents and presidents-elect at this "Conference for County Presidents," and we hope those present will inspire their local members to become more involved in the work of this Auxiliary. One of our important functions is to provide the opportunity to know other physician spouses as friends.

Meldon Laury
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At the request of the Impaired Physicians Committee of the Kansas Medical Society, space has been made available in the *Journal* for a section featuring articles relating to concerns and problems unique to the lifestyle of the physician. Articles may focus on communication, stress and distress, responsibilities to self, medical marriage, recreation and leisure, and related topics. Manuscripts or suggested topics and questions are solicited and should be submitted to:

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The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

Membership Categories

Certificate	$500—C
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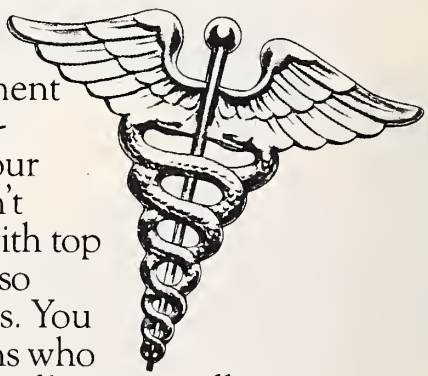
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(Continued on page 457)

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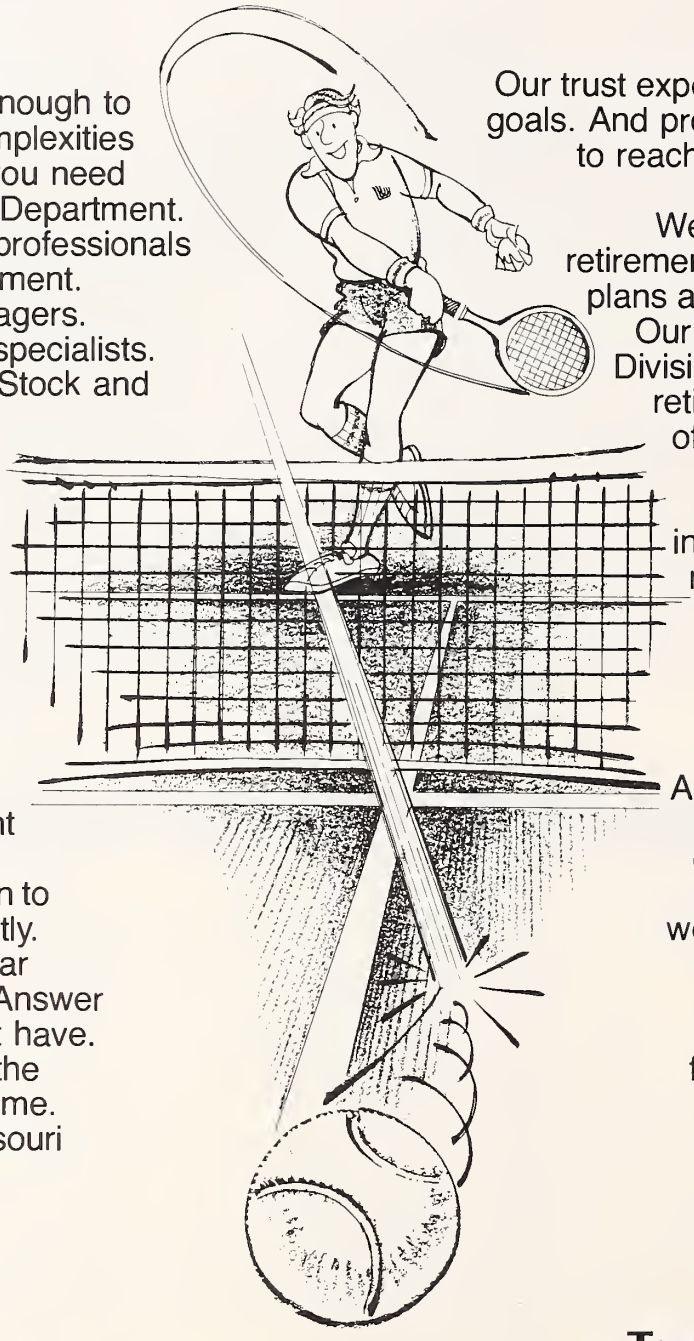
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Ninth Annual UKSM — Wichita Issue

The Dean's Letter — 1982

The University of Kansas School of Medicine – Wichita

WILLIAM J. REALS, M.D., *Wichita*

We are pleased to present this annual scientific portion of the September 1982 issue of *The Journal of the Kansas Medical Society*. A number of our faculty members have prepared papers of broad interest and of high scientific quality which reflect the work of our faculty in medical education in Kansas.

Phase I of the remodeling project of our UKSM-Wichita building is now complete. A new roof, new heating and cooling plant, new energy conserving windows, and decorative trim have been installed. Phase II is underway, and consists of complete interior remodeling with new offices and classroom space provided for educational activities. Phase III, the final portion of the $4.3 million project, will be completed in 1984.

Fifty medical students from the sophomore class come to Wichita in July each year to begin clinical training on this campus of UKSM. In addition to classes in the school building, they also attend numerous classes, conferences, and bedside teaching sessions at our four affiliated hospitals: Wesley Medical Center, The Veterans Administration Medical Center, St. Joseph Medical Center, and St. Francis Regional Medical Center. Without these cooperating hospitals, our school could not exist, and we are grateful for the many services they provide for us and for medical education. In addition, these allied hospitals provide settings for graduate medical education, including residencies in most of the major medical specialties. More than 450 physicians staff volunteer teaching appointments in addition to the 30 full-time and 80 part-time faculty people. Our staff of 135 people maintains the building, supports the educational efforts, and assists in the treatment of patients in our out-patient clinic.

Our school is community-based — that is, it utilizes community resources of the Wichita area — and nationally is the most successful example of this new concept of a university without walls. Much credit is due to the medical and hospital community in Wichita for their continuing support and interest in medical education. We also have enjoyed outstanding support from the University of Kansas Medical Center in Kansas City and the University of Kansas in Lawrence.

Our student body, faculty, and staff join me in expressing gratitude to all who have made our school a success. We pledge to continue to be an important element in providing health care to the citizens of Kansas.

Departmental Reports

Pediatrics

GERARD VAN LEEUWEN, M.D.

Richard A. Guthrie, M.D., Professor and Chairman of the Department, resigned in September 1981 to prepare for a sabbatical leave to be taken at the University of Florida, Miami. Dr. Guthrie and the Diabetes Team members are conducting the following research projects:

The Biosynthetic Human Insulin Study
The Nordisk Pork Insulin Study
The Relation of Pumps with Microvascular Control
The Changes in Pregnancy (Fetal Outcome-Diabetes)
The Time Curve of Insulin as Related to the Biostator Glucose Monitor

Other research in the Department includes the following:

Donald S. Seery, P.A.: Cost containment related to the biostator glucose monitor.

Diana Speelman, M.Ed., Counselor: Youth groups (care related to control).

Judy Freisen, R.D., Dietitian: Dental caries *vs* control.

Olga A. Tatpati, M.D. (who will complete a fellowship in Pediatric Endocrinology/Metabolism): Foot changes in diabetes.

Kirby Conley, R.N., Research Nurse: Insulin quality control with the biostator glucose monitor.

Joyce Dinkler, R.D., Dietitian: Points *vs* the Exchange System.

Debbie Hinnen, R.N., Diabetes Nurse Educator; Lindy Childs, R.N., Research Nurse; and Diana Speelman, M.Ed., counselor: Insulin pump screening — Locus of Control.

Diana Guthrie, R.N., will also be taking a one year sabbatical leave to study at the University of Florida, Miami, and will complete work for Ph.D. candidacy in counseling.

During the past year, the Diabetes Team moved from the Medical School to the St. Francis Regional Medical Center Pavilion where space was allocated for the Kansas Regional Diabetes Center and its administrative offices. A self-care unit was donated by the hospital for non-acute diabetic patients who require hospitalization, and this has greatly enhanced the Diabetes Program.

The Department grew slightly during the year with the addition half time of Margaret Wilson, M.D., who completed her residency with us in July 1981.

Our Residency Program had eight residents this past year, and will have 11 this year — five first year and three each in the second and third year programs. All of these residents are graduates of our UKSM-Wichita school except for one each from Kansas City Osteopathic School and the University of Nevada School of Medicine-Reno.

Patsy Barker, M.D., who completed her residency here in June, remains at St. Francis Regional Medical Center. Two others completed our training program this past year: Linda Ozaki, M.D., will spend two years in public health service, and John Jambura, M.D., is establishing private practice in Boise, Idaho.

Several people were promoted in Pediatrics during the year. Gerard J. Van Leeuwen, M.D., received tenure; Diana Guthrie, R.N., was promoted to Associate Professor with tenure; Richard G. Hoffman, Ph.D., was promoted to Assistant Professor; and Russell A. Nelson, M.D., was promoted to full professorship.

Family and Community Medicine

E. P. DONATELLE, M.D., *Chairman*

The Department of Family and Community Medicine at UKSM-Wichita has progressed to an active stage of development. New concepts are being introduced, and old ideas are being refined into more efficient, cost effective, and acceptable forms for implementation and evaluation of education for family practice at the undergraduate, graduate, and postgraduate levels. These efforts have included a careful assessment of past faculty performance and the addition of new faculty to our department staff.

Currently the department faculty consists of 12 full time, 28 part time, and 57 volunteer physicians from Sedgwick and neighboring counties.

The administrative structure now includes an Administrative Assistant, Vice Chairman of Undergraduate Education in Family Practice, and Heads of Divisions of Community Medicine, Human Behavior in Health Care, Postgraduate Studies (residencies and fellowships), and Research and Development. Because of the rapidly enlarging numbers of elderly, concepts for infusion of education for Geriatric care into both the undergraduate and graduate curricula are currently being developed in the Division of Community Medicine.

Since we have now completed the transition from a three to four year curriculum at the Wichita campus, we have assumed responsibility for 100 student months of curricular time for the purpose of teaching Family Practice. This is divided into a one month Clerkship during the Junior year and a one month Rural Preceptorship during the Senior year.

Our third year Clerkship has been completely revised to include basic concepts of Family Practice in a more acceptable patient oriented and patient involvement modular protocol. Students in the third year gain experience in the Wichita urban environment utilizing the family practice center for residency education at St. Francis Regional Medical Center, St. Joseph Medical Center, Wesley Medical Center, and private physicians' offices. A one month Rural Preceptorship experience has also been revitalized in its ultimate goals and the objectives to achieve these goals. There are now five rural teaching sites — one each in Harper, Lyons, Greensburg, McPherson, and Sterling — and we continue to upgrade our efforts to provide our students with a model of family practice care. Active involvement by full time department faculty ensures a closer relationship for improvement of the program, faculty enrichment, and evaluation of outcomes.

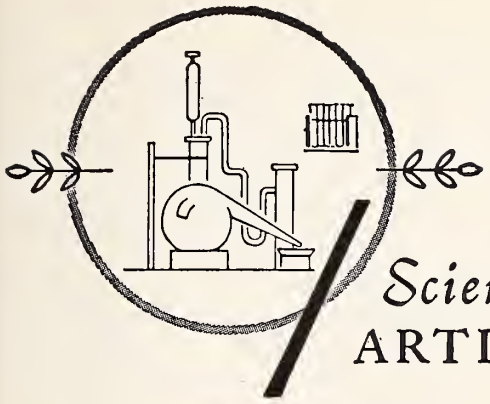
Graduate Education and training for family practice under the guidance of the Department of Family and Community Medicine is invested in four divisional residency programs. These programs are located at St. Joseph Medical Center, Wesley Medical Center, and St. Francis Regional Medical Center in Wichita, and the Smoky Hill Family Practice Center in Salina. Seventy-two residents are enrolled in all divisional programs during the current resident year which began in July. Student recruitment for all five Family Practice Residencies has been successful.

The Division of Continuing Medical Education (CME) has successfully launched the first of a continuum of major educational postgraduate programs for practicing physicians. Department faculty have also participated in lecture series provided for rural physicians by the Department of Continuing Medical Education, UKSM-Wichita.

A Presidential Award of $7,500, received by the Department through President Ernie J. Chaney, M.D., Belleville, American Academy of Family Physicians, funded development of a special fellowship for selected Kansas physicians. Philanthropic donations to the Department honoring the late James W. Shaw, M.D., a Wichita family practitioner for 50 years, have enabled the Department to establish the James W. Shaw Visiting Professorship Fund.

The Division of Community Medicine is being developed and a faculty person to head that division is currently being recruited. Research has been initiated in the study of assessment techniques designed to provide information regarding individuals in families at risk for health related problems. Studies are currently being conducted to determine how effective programs in Geriatric Medicine can be introduced in both the undergraduate and graduate levels at UKSM-Wichita. Perinatal death in Sedgwick county will be studied next year. It is hoped that active involvement in community research programs can be generated as a Division of Community Medicine becomes a more active part of Departmental functions. Through the guidance of the Department, the Kansas Academy of Family Physicians is developing a Family Practice Club as a continuum of a similar organization at the Kansas City campus. This will represent a new activity for medical students at the Wichita campus. A close liaison has been established between the Kansas Academy of Family Physicians and the Department of Family and Community Medicine here in Wichita.

Harmonious and cooperative relationships have been maintained with other departments, hospitals, and the community. It is encouraging to view the rapid developments at the UKSM-Wichita campus.



Exercise Training Program

Clinical Guidelines

E. P. DONATELLE, M.D.,* *Wichita*

EXERCISE in America has finally become socially accepted as the "in" thing, but there are valid physiological and psychological reasons for entering into a structured exercise program. Reasons vary depending on age, sex, and desired anticipated outcomes of the individuals concerned. In the younger age group, including those of college level, persons of both sexes express exercise in competitive sports, for fun, for body building, and for social prestige among their peers. In professional sports, in addition to the above objectives, an element of economics is a motivating force for continued training and excellence in the realm of exercise. For the middle-aged and the elderly, exercise training is a useful therapeutic intervention for prevention of disease of the cardiovascular system by achieving cardiovascular muscular and metabolic adaptation to increased aerobic work stimulus. The value of exercise is also achieved in physical and psychological treatment and rehabilitation of disease, and in health maintenance and wellness programs.[1] Exercise training, regardless of the reason, is a useful, therapeutic tool for most normal untrained individuals.

People choose to exercise for different reasons at different times in their lives[2-7] *(Table I)*. There are criteria that must be met to insure benefits while exercising[2, 3, 6] *(Table II)*.

* Professor and Chairman, Department of Family and Community Medicine, The University of Kansas School of Medicine-Wichita.

Address reprint requests to Dr. Donatelle, UKSM-Wichita, 1001 No. Minneapolis, Wichita KS 67214.

A clinical prescription for exercise requires a rational selection of a specific type of exercise to meet individual needs. The prescription combines an evaluation of an individual and his/her understanding of the basic exercise physiology as well as anticipated response outcomes to the training process.

Exercise has been accepted by the American public as a healthy modification of our way of life. Inappropriate involvement in exercise programs has resulted in failure to achieve anticipated goals, dampened enthusiasm, discontinuance, and — in some instances — serious physical as well as psychological injury. The information presented is designed to alert the prescribing physician and those involved to the benefits and dangers of exercise activities.

Clinical Evaluation Goal

Once an individual has decided on a desired type of exercise, s/he must have a medical examination to determine suitability for exercise training and to identify all risk factors which may require modification or limitation of his/her selected program. The extensiveness of medical screening will vary according to the four general groups encountered.[2, 3, 6, 8]

Group I: Under Age 30 Years. Any person under the age of 30 years, who is normally active without major cardiovascular risk factors previously deter-

TABLE I
REASONS FOR AN EXERCISE PROGRAM

- Social acceptability ("it's the in-thing")
- Personal self-image (Mr. America/Ms America)
- Preparation for competitive sports
- Requirement for physically demanding jobs
- Prevention of arteriovascular disease (Aerobic exercise increases in myocardial efficiencies and coronary collateral vascularization)
- Peripheral blood distribution and return
- Increased high density lipoproteins
- Increased vital lung capacity
- Improved thyroid function
- Improved growth hormone production
- Decreased arterial blood pressure both systolic and diastolic in a normal individual
- Reduced obesity, improved glucose tolerance, reduced triglyceride and cholesterol levels
- Reduced bone demineralization
- Reduced platelet aggregation
- Improved pulmonary ventilation rate
- Reduced psychic stress

mined by evaluation, can feel free to enter into an exercise program without further evaluation.

Group II: 30-40 Years. Apparently healthy 30-40-year-olds, who are normally active without major cardiovascular risk factors, may be adequately evaluated with a simple, pertinent medical history and systems-oriented physical examination. These individuals, particularly males, should have a resting electrocardiogram (ECG) and basic laboratory studies such as complete blood count (CBC), determination of fasting blood glucose, cholesterol, triglyceride levels, high density lipoproteins, and urinalysis. These studies should be repeated every two

TABLE II
CRITERIA FOR EXERCISE BENEFITS

- There must be an overload on muscle — skeletal and heart tissue.
- The progression of the exercise must be gradual and continuous.
- All exercises must be beneficial.
- Exercise must build both strength and muscular endurance.
- Exercise must be part of a more comprehensive treatment program including:
 —supervision
 —periodic evaluation
 —diet
 —medications when indicated
 —attention to physiological and social adjustment

TABLE III
CLINICAL APPLICATIONS OF AEROBIC EXERCISE*
THE EXERCISE PRESCRIPTION

How to Determine the Initial Recommendation for Adult Aerobic Exercise

Step 1. Estimate Maximum Aerobic Capacity:

Age Group	*Maximum Aerobic Capacity of Sedentary Adult (METS)†* *Men*	*Women*	*Maximum Pulse Rate*
21-30	12	10	195
31-40	11	9	191
41-50	10.5	8	187
51-60	9.5	7.5	182
61-70	8.5	6.0	178
71-80	6.0	5.5	174

Step 2. Determine 60% of Maximum Aerobic Capacity and 70% Maximum Pulse Rate

Step 3. Determine Appropriate Activity

MET Range	*Activity Examples*
2-3	Strolling; shuffleboard; bowling
4-5	Walk 4 mph; cycle 8 mph; golf, light carpentry
6-7	Walk-jog 5 mph; cycle 10 mph; badminton, splitting wood
8-9	Jog-run 6 mph; downhill skiing; swimming; tennis
10-11	Jog-run 7 mph; handball; racketball; heavy shoveling
12 plus	Run 8-10 mph; vigorous competitive sports

Step 4. Write Exercise Prescription
Activity
Intensity
Duration
Frequency
Precautions

* A scientific exhibit presented by the Department of Family and Community Medicine, Bowman Gray School of Medicine, Winston Salem NC 27103.[6] Used with permission.

† 1 MET is the energy expenditure at rest, equivalent to approximately 3.5 ml O_2/kg body weight per minute.

to three years in all who are established in an exercise program.

The necessity for graded exercise testing in this age group remains controversial in spite of the existing guidelines.[9] It has been the general experience of exercise physiologists that treadmill exercise testing is unnecessary for the apparently healthy 30-40-year-old who is active in recreational sports requiring peak energy expenditures of 8-10 METs (1 MET = energy expenditure at rest, equivalent to approximately 3.5 ml O_2/kg body weight/min).

Group III: Over 40 Years. Asymptomatic patients over 40 years of age and all patients with major

TABLE IV
APPROXIMATE METABOLIC COST OF ACTIVITIES*

	Occupational	*Recreational*
1½-2 METs†	Desk work	Standing
4-7 ml O_2/min/kg	Auto driving	Walking (strolling 1.6 km or 1 mile/hr)
2-2½ kcal/min (70 kg person)	Typing	Flying,‡ motorcycling‡
	Electric calculating machine operation	Playing cards‡
		Sewing, knitting
2-3 METs	Auto repair	Level walking (3¼ km or 2 miles/hr)
7-11 ml O_2/min/kg	Radio, TV repair	Level bicycling (8 km or 5 mile/hr)
2½-4 kcal/min (70 kg person)	Janitorial work	Riding lawn mower
	Typing, manual	Billards, bowling
	Bartending	Skeet,‡ shuffleboard
		Woodworking (light)
		Powerboat driving‡
		Golf (power cart)
		Canoeing (4 km or 2½ miles/hr)
		Horseback riding (walk)
		Playing piano and many musical instruments
3-4 METs	Bricklaying, plastering	Walking (5 km or 3 miles/hr)
11-14 ml O_2/min/kg	Wheelbarrow (220 kg or 100 lb load)	Cycling (10 km or 6 miles/hr)
4-5 kcal/min (70 kg person)		Horseshoe pitching
	Machine assembly	Volleyball (6 man noncompetitive)
	Trailer-truck in traffic	Golf (pulling bag cart)
	Welding (moderate load)	Archery
	Cleaning windows	Sailing (handling small boat)
		Fly fishing (standing with waders)
		Horseback (sitting to trot)
		Badminton (social doubles)
		Pushing light power mower
		Energetic musician
4-5 METs 14-18 ml O_2/min/kg	Painting, masonry	Walking (5½ km or 3½ miles/hr)
	Paperhanging	Cycling (13 km or 8 miles/hr)
5-6 kcal/min (70 kg person)	Light carpentry	Table tennis
		Golf (carrying clubs)
		Dancing (foxtrot)
		Badminton (singles)
		Tennis (doubles)
		Raking leaves
		Hoeing
		Many calisthenics
5-6 METs	Digging garden	Walking (6½ km or 4 miles/hr)
18-21 ml O_2/min/kg	Shoveling light earth	Cycling (16 km or 10 miles/hr)
6-7 kcal/min (70 kg person)		Canoeing (6½ km or 4 miles/hr)
		Horseback (''posting'' to trot)
		Stream fishing (walking in light current in waders)
		Ice or rollerskating (15 km or 9 miles/hr)
6-7 METs	Shoveling 10/min (22 kg or 10 lbs)	Walking (8 km or 5 miles/hr)
21-25 ml O_2/min/kg		Cycling (17½ km or 11 miles/hr)
7-8 kcal/min (70 kg person)		Badminton (competitive)
		Tennis (singles)
		Splitting wood
		Snow shoveling
		Hand lawn-mowing
		Folk (square) dancing
		Light downhill skiing
		Ski touring (4 km or 2½ miles/hr) (loose snow)
		Water skiing

(Continued on next page)

TABLE IV — *Continued*

7-8 METs 25-28 Ml O_2/min/kg 8-10 kcal/min (70 kg person)	Digging ditches Carrying 175 kg or 80 lbs Sawing hardwood	Jogging (8 km or 5 miles/hr) Cycling (19 km or 12 miles/hr) Horseback (gallop) Vigorous downhill skiing Basketball Mountain climbing Ice hockey Canoeing (8 km or 5 miles/hr) Touch football Paddleball
8-9 METs 28-32 ml O_2/min/kg 10-11 kcal/min (70 kg person)	Shoveling 10/min (31 kg or 14 lbs)	Running (9 km or 5½ miles/hr) Cycling (21 km or 13 miles/hr) Ski touring (6½ km or 4 miles/hr) (loose snow) Squash racquets (social) Handball (social) Fencing Basketball (vigorous)
10 plus METs 32 plus ml O_2/min/kg 11 plus kcal/min (70 kg person)	Shoveling 10/min (35 kg or 16 lbs)	Running: 6 mph = 10 METs 7 mph = 11½ METs 8 mph = 13½ METs 9 mph = 15 METs 10 mph = 17 METs Ski touring (8+ km or 5+ miles/hr) (loose snow) Handball (competitive) Squash (competitive)

* Includes resting metabolic needs.

† 1 MET is the energy expenditure at rest, equivalent to approx. 3.5 ml O_2/kg body weight/minute.

‡ A major excess metabolic increase may occur due to excitement, anxiety, or impatience in some of these activities, and a physician must assess his patient's psychological reactivity.

cardiovascular risk factors or suspected cardiovascular disease should have a thorough screening including a medical historical profile, physical examination, laboratory study, resting electrocardiogram, and graded exercise stress test. The medical history and physical examination should concentrate on cardiovascular, pulmonary, metabolic, and muscular skeletal functions with special attention to the efforts related to symptoms of dyspnea, chest pain, dizziness, weakness, and claudication.

Basic laboratory studies should include an attempt to identify biochemical risk factors. Routine chest x-rays and spirometry are suggested for patients with a questionable history of pulmonary disease. The risk factors of significance are: obesity, hypertension, diabetes, previous injury, acute or chronic pulmonary disease, hyperlipoproteinemia, and inherited risk factors related to cardiovascular disorders.

Group IV: Pre-existing Disorders. Patients with established cardiovascular disease or other chronic disorders must be carefully evaluated for potential problems or limitation. Medications may alter cardiovascular response (beta blockers, ganglionic blockers, vasodilators — all related to treatment of hypertension) or muscle function. Potassium depleting diuretics must be considered when determining clinical status for exercise training.

Following myocardial infarction or coronary bypass, patients should proceed through three phases of cardiovascular rehabilitation: Phase I — in-hospital period of training immediately following the incurred insult; Phase II — provides progressive exercise rehabilitation under observation; and Phase III — exercise training which may be conducted in supervised adult fitness cardio-rehabilitation programs.[4, 5, 9, 10]

Prerequisites for Exercise Prescription

Objective medical screening will determine which patients are suitable candidates for exercise training and will identify all risk factors which may modify or limit an exercise program.[6]

Evaluative procedures for apparently healthy persons 30-40 years of age should include: history (patient historical medical profile), physical examination, resting electrocardiogram, and laboratory studies to include CBC, fasting glucose, cholesterol triglycerides, high density lipoproteins, and urinalysis.

Asymptomatic patients over 40 years of age and all patients with major cardiovascular risks or suspected cardiovascular disease should be evaluated as above with the addition of graduated exercise stress testing and chest x-ray and spirometry.

The exercise program for the patient with established cardiovascular disease or other chronic disorders should be managed under the direct supervision of a physician in a rehabilitation center. Evaluation for these patients should include a complete history and physical examination[4, 6, 9, 10] in addition to the evaluative procedures previously listed.

TABLE V
PROTOCOL FOR EXERCISE PRESCRIPTION

- Anyone who wishes to begin a program of planned physical exercise should have an appropriate medical evaluation considering age, sex, physical capacity, and health.
- All exercises initiated should be modified in a starter program protocol prior to participation in a vigorous physical activity program.
- Kinetic exercises such as walking, jogging, swimming, and cycling are recommended as opposed to isometrics or static exercises such as weight lifting.
- Effective physical training relates to the type of activity, its intensity, duration, and frequency. Serious reservations exist concerning the advisability of high intensity activity as a means of maintaining a desired level of fitness.
- Responses during exercise which should lead to medical review include the following:
 —Any chest pain or pain referred to the teeth, arm, jaw, or ear.
 —Syncope, light headedness, or dizziness following exercise.
 —Irregular heart rate following exercise.
 —Persistent fatigue.
 —Unusual weight loss.
 —Muscular skeletal problems aggravated by exercise.
 —Nausea, vomiting, or feeling of malaise.
 —Failure of pulse rate to recover below 140 level within 5 minutes following exercise.
- A heart rate following cessation of exercise should normally show recovery tendencies returning to 120 level within approximately 3 minutes. If higher persists, this may be an indication of overwork.
- An exercise program should be composed of three parts including:
 —warmup
 —work phase
 —cool out or tapering off
- Subjects who have been sedentary should exercise extreme caution prior to undertaking an exercise program which includes vigorous physical activity.
- In terms of heart rate for men and women of ages 35-55 years, light, moderate, and vigorous physical activity may be defined as follows:
 —light — 100 to 120/min
 —moderate — 120 to 140/min
 —vigorous — 140/min and up
- Breath holding or the VALSALVA maneuver should not be included in any portion of the activity program.
- Environmental factors such as heat, humidity, and elevation should be considered when participating in a physical exercise program.

The Exercise Prescription

A suitable medical evaluation provides the basis for selection of an appropriate exercise corresponding to an individual's desire and capacity. This can easily be determined by utilization of a protocol entitled "Clinical Application of Aerobic Exercise" established in the Department of Family and Community Medicine, Bowman Gray School of Medicine *(Tables III* and *IV)*. It should be noted that although selection of the type of exercise corresponding to one's age can be made by anyone following the stated guidelines, the *activity, intensity, duration, frequency,* and *precautions* for each individual's exercise must be the responsibility of his/her physician.

Whether or not an individual elects to enter into an exercise program or is so advised by friends or physicians, s/he must be made aware of the proper protocol and precautions to follow that will minimize the harmful effects of this potent prescribed medicine[2, 3, 6, 8] *(Table V)*.

Summary

During the past decade, exercise has been accepted by the American public as a healthy modification in our way of life. Effects of structured, disciplined, forms of exercise programs are beginning to reflect themselves in lowered incidents of cardiovascular disease, improved health maintenance, relief of stress, and general improvement in the quality of life.

Although exercise has many beneficial effects, it can also produce damaging physiological results if not appropriately prescribed for the individual concerned. These guidelines have been presented for physicians and others who seek to prescribe exercise training programs for patients or associates.

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Bipolar Affective Disorder

Report of a Case Treated With Carbamazepine (Tegretol)

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CONTROL AND PROPHYLAXIS of bipolar affective disease with the anticonvulsant carbamazepine (Tegretol) has been documented in Japanese literature for several years.[1-3] The original clinical trials of carbamazepine reported a "markedly effective" or "effective" antimanic effect in 42 per cent of patients with bipolar affective disease.[1] Successful prophylaxis of further manic and depressive episodes was reported in 73 per cent of patients. However, the interpretation of these results is difficult, because of the concomittant use of major tranquilizers and tricyclic antidepressants.

The same group of investigators found that the antimanic effect of carbamazepine was clinically superior, but not significantly different statistically, to that of chlorpromazine,[2] and the prophylactic effectiveness of carbamazepine was later substantiated in a double-blind comparison of carbamazepine and placebo.[3]

Recently, a double-blind placebo control design study done at the NIMH using patients with primary affective or schizoaffective illness reported that 70 per cent of the patients had an antimanic, antidepressive, or prophylactic response.[4] Interestingly, four of the patients with a history of nonresponse to lithium responded favorably to carbamazepine. One patient who did not respond to carbamazepine later responded well to lithium. This observation lends support to the speculation that carbamazepine may be effective for some lithium nonresponders and vice-versa.

Ballenger and Post conducted a double-blind placebo controlled study which further demonstrated the effect of carbamazepine in both mania and depression, and numerous additional reports are forthcoming.[5]

Because of the above reports, which began to appear in the late 1970s, the author decided to use carbamazepine to treat individuals with bipolar affective disorders who did not respond to lithium carbonate in order to avoid the morbidity associated with other neuroleptics. The following case illustrates the successful outcome in one such instance.

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Clinical trials of carbamazepine have indicated its usefulness in treatment of manic and depressive episodes in patients with bipolar affective disorders. The seriously impaired patient reported here was uncontrolled by other medications, but has functioned normally for several years on carbamazepine therapy.

Case Report

A 24-year-old white male college student first sought psychiatric treatment on May 31, 1977. His history at that time revealed the insidious onset of psychosis which had gradually worsened during the preceding 12 months. Initially, the patient began to avoid friends. This progressed until the date he was first seen. A that time, he was delusional and withdrawn to the point that he would not leave his apartment at college, kept his blinds closed at all times, neglected his personal hygiene, and quit attending classes. He spent his time either pacing or sleeping.

Past medical history was not remarkable except for a minor head injury without unconsciousnss which was incurred in an auto accident four years previously. Family history was negative for psychiatric or neurological illness. Laboratory evaluation at that time consisted of SMA-18, urinalysis, complete blood count, venereal disease research laboratory testing, thyroid function studies, chest x-rays, and EEG; results were all within normal limits. Physical examination was not remarkable. Mental status examination revealed a flat affect except for occasional inappropriate laughter, apparent auditory hallucinations, marked suspiciousness, loose associations, and considerable anxiety.

At the time the patient was initially seen, a diagnosis of schizophrenia, paranoid type, was considered appropriate. He was hospitalized for four

days during which a medical evaluation was done and treatment with haloperidol, 2 mg four times per day, was begun. The psychosis responded promptly to treatment with haloperidol, but the patient developed incapacitating extrapyramidal symptoms. He was again hospitalized on August 3, and his medication was changed to thiothixene, 5 mg and benzotropine, 1 mg three times per day. This resulted in marked reduction of extrapyramidal symptoms. During the next six months, the thiothixene dosage was gradually decreased and it was found that the psychosis remained in remission without neuroleptics.

However, in July 1978, the patient returned for a routine followup visit in a hyperactive state with pressured speech, flight of ideas, grandiose delusions, and irritability. A brief attempt was made to treat the patient on an outpatient basis with lithium carbonate, but on July 7 he was again hospitalized. Treatment with lithium carbonate 1500 mg/day; perphenazine, 24 mg/day; and benzotropine, 2 mg two times per day resulted in prompt remission of delusional thinking, but the patient developed moderate extrapyramidal symptoms and continued to be hypomanic.

During the following 38 days the patient cycled from a hypomanic state to a withdrawn state similar to the state he was in during his first admission. Attempts were made to treat this by changing neuroleptic medications from perphenazine to thiothixene and finally by the addition of imipramine in low doses. The latter resulted in the patient developing a bizarre agitated psychotic state.

On June 7, 1979, the patient was again hospitalized because of continued cycles from severe withdrawal to hypomania. This occurred even though serum lithim levels were adequate and the patient was compliant with taking thiothixene, 15 mg/day. Symptoms had been such that the patient had required custodial care since July, 1978.

During this admission, the patient was started on carbamazepine, 200 mg two times per day which was progressively increased to 200 mg four times per day. The thiothixene was gradually discontinued, but lithium carbonate, 1500 mg/day was continued. The patient's emotional state rapidly normalized and he was discharged on June 22, 1979.

Since that time, the patient has functioned normally, including full-time employment, except for two episodes of insomnia lasting seven days and two days, respectively. The first period of insomnia was temporally correlated with a drop in the serum carbamazepine level 30 days after the patient had begun taking that medication. He returned to his normal sleep pattern with an increase in carbamazepine dosage to 400 mg three times per day. The second episode of insomnia remitted without treatment.

Discussion

The deleterious side effects of most neuroleptics — such as psychomotor retardation, pseudoparkinsonism (or other extrapyramidal symptoms), sedation, and decreased ability to learn — are well recognized. Although neuroleptics can be quite efficacious in the control of psychotic symptoms, the side effects may be sufficient to prevent return of the patient to full functioning. In addition, when treating bipolar affective disease, neuroleptics have little, if any, effect on the depressive components of the disease. The use of neuroleptics has also been related to the development of dyskinesia which may be as disabling as the illness being treated.

The predominant side effects of carbamazepine are sedation and a decrease in leukocytes. Only rarely is aplastic anemia seen in conjunction with carbamazepine administration. Patients being treated with carbamazepine do not complain of psychomotor slowing, and the drug does not impair learning.[6] Extensive use of carbamazepine for treatment of seizure disorders and paroxysmal pain syndromes has demonstrated its safety.

All reports have indicated a rapid onset of the antimanic effects of carbamazepine, usually within a matter of days. It does not cause an increase in urinary output or tremor.

The effective antimanic dosage of carbamazepine is not clear. This author has maintained the therapeutic anticonvulsant blood level in all patients, and has seen a return of symptomatology in a number of patients when the blood level has fallen below the therapeutic range. However, controlled studies have not correlated blood level with efficacy.[2, 3]

The mechanism of action of carbamazepine is not known. It appears to have a direct effect on the vasopressin binding site, decreases cerebrospinal fluid somatostatin, blocks norepinephrine re-uptake, and decreases gamma-aminobutyric acid turnover in animals.[7] It is hypothesized that the effect of carbamazepine on affective disorders is related to its ability to stabilize temporal lobe and limbic dysfunction.[7]

Summary

Carbamazepine appears to be effective in the treatment and prophylaxis of bipolar affective disorder, including some patients with whom lithium carbonate was ineffective. This is especially note-

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Page Kidney

An Unusual Cause of Curable Hypertension

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An animal model of pressor mediated hypertension due to chronic renal parenchymal compression was described by Irvine Page in 1939.[1] In this model — cellophane perinephritis — the kidney is encased in a wrapping of cellophane. Tissue reaction to the cellophane results in the formation of a constricting fibrocollagenous hull which develops around the kidney during a period of two to three weeks. In three to five weeks hypertension appears in the animal model. Removal of the involved kidney normalizes the blood pressure.

Hypertension is mediated through the renin-angiotensin system. Compression by the constricting fibrocollagenous hull results in regional hypoperfusion of the kidney, and release of renin and subsequent generation of angiotensin II. Angiotensin II, in addition to being a potent vasoconstrictor, stimulates the release of aldosterone from the adrenal gland. Activation of these humoral limbs results in hypertension.

In this paper we discuss two hypertensive patients who were found to demonstrate the anatomic and physiologic abnormalities corresponding to the experimental model, cellophane perinephritis.

Case One

A 25-year-old white male was discovered to have asymptomatic hypertension two years after sustaining left flank trauma during a football game. Blood pressure was 174/102 mmHg on his initial visit. Results of physical examination were normal. Laboratory examination demonstrated a mild erythrocystosis with hemoglobin, 17.3 gm/dl and hematocrit, 49.6%. Renal function was normal with a creatinine clearance of 110 ml/min.

Renal arteriography showed no abnormality of the main renal arteries. The left kidney was found to be displaced anteriorly by a large avascular mass, measuring 15 × 8 cm. The blood vessels of the left kidney were stretched across this mass and calcifications could be seen in the anterior portion of the mass.

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Segmental renal vein renin determinations in this patient demonstrated a significant increase in renin coming from the upper pole of the left kidney *(Table I)*.

Page kidney, an unusual variant of pressor hypertension, most commonly results from tumor or trauma that impairs the kidney's vascular function. In carefully selected cases, surgical treatment may completely ameliorate hypertension or simplify its control.

Surgical exploration was accomplished through a flank incision. A chronic subcapsular hematoma was found to be adherent to the parenchyma of the left kidney *(Figure 1)*. A left nephrectomy was performed. Following surgery the patient's blood pressure normalized with no need for antihypertensive medications.

Case Two

A 44-year-old white male incidentally was discovered to have hypertension during management of chronic pancreatitis. Initial arm blood pressure reading was 190/110 mmHg, and values as high as 230/130 mmHg were recorded during a period of 18 months followup as medical control was attempted.

Physical examination uncovered a continuous bruit present in the left hemiabdomen. Tenderness was present on palpation in the left upper quadrant, and a vaguely outlined mass appeared to be present.

TABLE I

	Renal Vein Renin (ng/ml/hr)			
	Patient One		*Patient Two*	
Sample Site	*Right*	*Left*	*Right*	*Left*
Upper kidney	—	3.7	—	18
Lower kidney	—	2.2	—	18.5
Main vein	1.6	2.2	25	17
Vena cava	1.6	—	19	—



Figure 1: Left kidney *(Case One)* showing chronic subcapsular hematoma.

Laboratory examination indicated a normal serum creatinine of 1 mg/dl. The creatinine clearance was moderately reduced at 69 ml/min, indirectly reflecting the patient's chronic malnutrition from pancreatitis.

Renal arteriography *(Figure 2)* was performed and demonstrated normal renal arteries. Selective renal arteriography demonstrated a large mass in the superior, lateral aspect of the left kidney. The mass was seen to displace the intrarenal blood vessels as it impinged on the kidney.

Segmental renal vein renin determinations demonstrated marked elevation in samples obtained from both kidneys as well as from the inferior vena cava *(Table I)*. The vascular system was flooded with renin.

Surgical exploration was accomplished through a transverse upper abdominal incision. An organized abscess of the tail of the pancreas was found to be



Figure 2: Renal arteriogram *(Case Two)* demonstrating large mass in the superior lateral aspect of the left kidney.

adherent to the capsule of the left kidney, resulting in parenchymal compression. A distal pancreatectomy, splenectomy, and left nephrectomy were performed.

Following surgery the peripheral renin fell to 3.7 ng/ml/hr. The blood pressure fell, but did not completely normalize as in *Case One*. Blood pressure control, however, required much less medication and was easier to achieve following surgical removal of the chronically compressed left kidney.

Discussion

The majority of patients with hypertension will have no identifiable cause for their high blood pressure, and are described as having essential hypertension. Approximately 5 per cent of the hypertensive population will have an identifiable cause for high blood pressure, and these disorders most commonly involve the renin-angiotensin II-aldosterone axis. While uncommon, disorders of the renin-angiotensin II-aldosterone axis are often amenable to surgical cure, and require vigilance on the part of the practitioner lest they be overlooked.

Renin is a proteolytic enzyme elaborated in the juxtaglomerular cells of the kidney. Renin acts upon an alpha globulin substrate from the liver to form the decapeptide angiotensin I. Angiotensin I in turn is converted to the octapeptide angiotensin II by an enzyme localized principally in the lung. Angiotensin II stimulates the release of aldosterone from the adrenal gland in addition to being a potent vasoconstrictor.

Abnormalities of the renin-angiotensin II-aldosterone axis comprise a spectrum of clinical hypertension, from purely volume mediated hyper-

tension (aldosterone) to pressor mediated hypertension (angiotensin II). An example of volume mediated hypertension is primary aldosteronism; an example of pressor mediated hypertension is renal artery stenosis. These two examples of extremes in the clinical spectrum of hypertension have been studied in laboratory animals. The classic model of pressor hypertension is the Goldblatt two-kidney preparation described in 1934. Less well known is the Goldblatt one-kidney preparation, a model of volume mediated hypertension. In the two-kidney Goldblatt preparation, one renal artery is clamped and the contralateral kidney is left untouched. This model most clearly simulates the human condition of renal artery stenosis. Page kidney is an extension of this classic model in that compression of the renal parenchyma, akin to clamping the renal artery, results in pressor mediated hypertension. At the other end of the spectrum is the one-kidney Goldblatt model in which one renal artery is clamped and the contralateral kidney is removed. Hypertension in this model is caused by salt and water retention and volume expansion. Renin elaboration by the kidney is suppressed.

In the two-kidney Goldblatt model and the Page kidney model, hypertension is initially pressor mediated by the elaboration of angiotensin II. With time, however, the elevated blood pressure is incapable of correction by administration of angiotensin II blockers. Improvement can be achieved only with salt restriction of diuretics. The initial pressor mediated hypertension is now, in addition, volume dependent.[2]

A pragmatic point can be made in human counterparts of these animal models. The earlier the diagnosis of hypertension is established the greater the chance for surgical cure. The longer the hypertension remains undiscovered, and the higher the chronic readings of blood pressure, the less the likelihood of complete surgical cure. The reason for this is arteriolar damage induced by the hypertension itself throughout the vascular tree.

The two patients discussed above most likely had a similar duration of hypertension of approximately two years. The higher blood pressure readings and older age of the latter patient, however, contributed to an inability to achieve complete surgical cure. Vascular damage due to hypertension was more widespread in this patient than in the earlier one.

While it is desirable to secure early surgical intervention in suspected cases of Page kidney, cure can still be accomplished late in the disease. Normotension has been achieved in some patients as long as 23 years after a traumatic incident.[3]

The two most common causes of Page kidney are trauma[4] and tumor compression of the kidney.[5] Following trauma bleeding may occur beneath the fibrous capsule of the kidney, resulting in compression of renal parenchyma by hematoma and subsequent hypertension. A variety of different tumors have been described as causing pressor hypertension. Compression by tumor mass most commonly involves the renal artery. The second patient, however, had direct renal compression by the pancreatic abscess resulting in renin production and hypertension. The two patients discussed in this paper are thus examples of Page kidney and not the Goldblatt two-kidney model.

The diagnosis of Page kidney begins with a careful history. The physician should attempt to discover whether or not flank trauma has occurred in the past and when it may have occurred. Recent onset of hypertension in a young patient should initiate suspicion of Page kidney. It is important to inquire about activities in contact sports and avocations such as trail bike riding. The two most important physical determinations are the level of blood pressure and the presence or absence of an abdominal bruit. A significant abdominal bruit is one that is present in both systole and diastole, and is transmitted toward the flank.

Diagnostic examination of an individual suspected of having Page kidney is designed to demonstrate both an anatomic and a physiologic abnormality. The renal arteriogram is the single best examination to demonstrate abnormalities of the renal vasculature. The desirability of preliminary screening examinations such as CAT scanning, intravenous pyelography, sonography, or isotope scanning will depend upon information elicited from the history and performance of a careful physical examination.

The demonstration of a physiologic abnormality corresponding to the anatomic lesion is best secured by performing split renal vein renin determinations. A common method involves calculating the renal vein renin ratio between the involved and the noninvolved kidneys. A value greater than 1.5 is predictive of surgical curability in patients with renal artery stenosis. More sophisticated criteria derived from renal renin measurements have been used to predict surgical cure. These maneuvers are designed to demonstrate suppression of renin in the noninvolved kidney, and the influence of renal blood flow on the level of renal vein renin. The amount of renin appearing in the renal vein blood will depend not only on how much is being elaborated, but also renal blood flow itself. Reduction in renal blood flow due to parenchymal compression could occur in

Page kidney, resulting in a falsely high renal vein renin value.[6]

An ancillary examination that may be performed in the individual with suspected Page kidney in order to demonstrate a physiologic lesion, is saralysin testing. Saralysin is a weak agonist of angiotensin II which theoretically would hold promise as a diagnostic and prognostic tool in patients being examined for Page kidney.

The decision as to whether to treat the patient with Page kidney medically or surgically will depend on many variables. If the patient is young and predictability studies show a good chance of surgical cure, surgery would be the best treatment. The older patient with multi-system disease who is not a good surgical candidate may be more safely managed on a medical regimen, and no diagnostic examinations should be performed in this type of patient. In the bulk of patients with Page kidney, surgical correction — usually involving nephrectomy — will be the most attractive therapy. Surgical correction should be considered in all patients demonstrated to have Page kidney. Attempts should be made to try to preserve as much renal tissue as possible and if correction of high blood pressure can be achieved by draining a perinephric hematoma for example, this is most desirable.

Pharmacologic management of hypertension in patients with Page kidney is not the best long term therapy, but may be necessary prior to surgery. Two agents — beta blockers and Captopril — may be tried because of their effectiveness in high renin hypertension.

Summary

In summary, we have presented two hypertensive patients who were found to demonstrate anatomic and physiologic abnormalities corresponding to the experimental model of renal compression, cellophane perinephritis, or Page kidney. Radiologic investigation uncovered unilateral renal disease in these patients which then prompted selective renal vein renin determinations. Renin determinations indicated the presence of a pressor hypertension mediated by the renin-angiotensin II-aldosterone system in both patients. Surgery resulted in complete amelioration of the hypertension in one patient and marked improvement, in terms of ease of control, in the second patient.

Page kidney is an unusual variant of pressor hypertension which must be considered in individuals who present with abrupt onset of hypertension at any age. Diagnostic evaluation includes methodology designed to demonstrate compression of the kidney, most commonly a result of tumor or trauma, and selective renal vein renin determinations to assess the functional consequences of parenchymal compression.

Awareness of Page kidney may permit surgical cure of hypertension.

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worthy because carbamazepine does not appear to have side effects that are as troublesome as other neuroleptics more commonly employed in the treatment of lithium nonresponders.

At this time, it appears that carbamazepine holds promise as an alternative treatment for patients with bipolar affective disorder who for one reason or another cannot be treated otherwise.

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Anaphylaxis to Succinylcholine

A Case Report and Discussion of Therapy

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ANAPHYLACTIC reactions to intravenous anesthetic agents are rare. True anaphylaxis to succinylcholine is especially rare with only 35-40 cases reported in the literature. Prompt recognition of anaphylaxis during surgery is essential to prevention of cardiovascular collapse. If recognzied and treated promptly and aggressively, full recovery without residual cerebral, cardiac, or other sequelae usually can be achieved.

Case Report

A 55-year-old white female with well-controlled hypertension presented for colon resection for divericulitis and diarrhea of one year's duration.

The patient had undergone surgery eight times previously at this same institution between 1975 and 1978 without any known anesthetic drug reactions, but had a past history of penicillin and sulfa drug allergies. Current medications included propranolol (Inderal) 20 mg three times per day and Aldactazide two times per day.

Results of laboratory tests included: hemoglobin, 12.8 gm/100 ml; and white blood count, 8800. Chest x-ray revealed no active disease. Results of urine analysis were within normal limits.

The day prior to surgery, the patient complained of itching and erythema of her hands, arms, and face following two days of intravenous cefamandole (Mandol).

Prior to surgery, her blood pressure was 110/80; pulse, 84; and temperature, 36.2C. Preoperative medications of meperidine, 25 mg; hydroxyzine (Vistaril), 50 mg; and glycopyrrolate (Robinul), 0.3 mg were given intramuscularly one hour prior to surgery.

Upon arrival in the operating room, blood pressure was 180/100 with a pulse of 130. The patient was induced with sodium thiopental, 400 mgm; d-Tubocurare (dTC), 6 mg; succinylcholine (SCH), 80 mg; fentanyl, 0.1 mg; and pancuronium, 5 mg.

Shortly after intubation, blood pressure was unobtainable by cuff. Electrocardiogram showed a regular rhythm with rate of 120 which dropped to 83 during the next few minutes. The patient developed a marked erythema on her upper torso.

Anaphylaxis to succinylcholine in the operating room is uncommon. When it occurs, prompt diagnosis and therapy are essential to prevent cardiac arrest and death. When recognized and treated promptly and aggressively, full recovery usually can be achieved.

Phenylephrine (Neosynephrine), 0.2 mg was administered resulting in a blood pessure of 240/120 and a pulse of 140. Propranolol, 0.25 mg and diphenhydramine (Benadryl), 50 mg were given as the erythema rapidly resolved. The surgical procedure was cancelled, and the patient had an uneventful post anesthetic course.

Three days later, the patient returned to surgery. Diphenhydramine, 50 mg and hydroxyzine, 50 mg were administered intramuscularly one hour preoperatively.

Induction was accomplished with thiopental, 250 mg intravenously slowly, followed by lidocaine, 75 mg and succinylcholine, 50 mg. Within minutes, erythema and flushing developed on the upper torso, and the blood pressure became non-palpable peripherally. Hydrocortisone, 100 mg and diphenhydramine, 50 mg were given intravenously. In addition hydrocortisone, 500 mg was added to the intravenous fluid bag. Ephedrine, 10 mg and phenylephrine, 0.2 mg quickly returned blood pressure to normal. Within 25 minutes, blood pressure stabilized at 120/80 mmHg, pulse at 70, and the erythema disappeared.

To achieve muscle relaxation for the procedure, a succinylcholine drip, 2 mg/ml, was started. Immediately, the erythema returned and blood pressure became unobtainable. The succinylcholine drip was

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stopped. A dopamine infusion quickly returned the blood pressure to 140/90 with a pulse of 120. The erythema again quickly subsided. The surgical procedure was performed without further incident. The patient had an uneventful post-operative course.

Discussion

Anaphylaxis following administration of succinylcholine is an uncommon occurrence. Risk factors include: a history of drug allergies, especially penicillin;[1] prior, repeated administration of the drug; and atopy.[2] This patient gave a history of penicillin allergy. She also had experienced two days of arm and facial pruritis coinciding with the intravenous administration of cefamandol. During all of her eight prior operations the patient received succinylcholine.

In addition to circulatory collapse, urticaria, erythema, and bronchospasm may be present during succinylcholine anaphylaxis. In a review of 13 cases by Moneret-Vautrin, *et al.*, cardiovascular collapse occurred in ten, bronchospasm in eight, generalized or upper body erythema in seven, and facial edema in two cases.[2] In this case, circulatory collapse and erythema occurred, but bronchospasm and urticaria were absent.

Anaphylaxis to methylparaben, a preservative found in succinylcholine solutions as well as in many other drugs and cosmetics, has been described.[3, 4] Methylparaben has been implicated as the true allergin in many of the alleged succinylcholine anaphylaxis reactions. However, in this particular case, a succinylcholine drip prepared from a powder free of preservative precipitated the same response.

In order to distinguish a true anaphylactic reaction with specific IgE antibodies to the muscle relaxant from an anaphylactoid reaction due to excessive histamine release, special tests must be performed.[1, 5] Skin testing, Prausnitz-Kustner testing for passive transfer of antibodies, human basophil degranulation testing, and IgE levels postoperatively[6] help to make this distinction.

Therapy

Once diagnosed, anaphylaxis must be treated promptly. Therapy should be aimed at maintaining good oxygenation and in providing adequate circulating volume. Airway management should include intubation to provide adequate ventilation.[7]

With the onset of edema, a decrease in circulating volume occurs. Therefore additional volume must be rapidly administered via a large intravenous catheter. Crystalloid is most frequently used for volume replacement.

Drug therapy should include epinephrine[8] 0.5-1.0 ml of 1 : 1000 dilution intravenously, intramuscularly, or subcutaneously as the drug of choice. Aminophylline is useful for bronchospasm. Corticosteroids and antihistamines are of questionable value in resuscitation since their onset of action is relatively slow.[9]

In this case, phenylephrine was the primary therapy for the hypotension. Since no bronchospasm was noted, therapy was directed at cardiovasular and peripheral vascular resuscitation for which phenylephrine proved very efficacious.

Conclusion

Anaphylaxis in the operating room requires prompt diagnosis and therapy to prevent cardiac arrest and death. Supportive and fluid therapy are the primary treatments essential for patient recovery. Whether a true anaphylaxis or an anaphylactoid reaction occurs is moot. What is essential is that the patient's cardiovascular and pulmonary status are quickly stabilized.

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Thyroglossal Cyst Carcinoma

A Case Report and Review of the Literature

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THYROGLOSSAL DUCT cyst is one of the commonly encountered benign cervical masses.[1] Carcinoma arising in a thyroglossal duct cyst is uncommon.[2, 3] Malignancy is seldom appreciated before surgery, and is usually not recognized until the permanent histology slides are reviewed. In 1927 Owen and Ingleby[4] reported the first case in the English literature of carcinoma arising in the thyroglossal duct tract. There are fewer than 100 reported cases so far that showed histologically documented cases of papillary carcinoma of the thyroglossal duct.[5-8] Reported here is a case seen recently at our institution; the available literature on the subject is reviewed.

Case Report

A 63-year-old male was admitted to the Wichita Veterans Administration Medical Center with the chief complaint of a mass in the midneck which had been discovered about four months prior to admission. He denied any difficulty with swallowing. On physical examination, the patient was found to have a midline neck mass which moved with swallowing and upon protrusion of the tongue. The mass was nontender, approximately 3 cm in diameter, and on palpatation appeared to be cystic. There was no lymphadenopathy in the area; the thyroid gland was not enlarged. The results of admission laboratory studies — which included complete blood count, urinalysis, and SMAC — were within normal limits. Chest x-ray showed emphysematous changes in the lungs and arteriosclerosis of the aorta without gross cardiomegaly. Electrocardiogram was normal. Thyroid scan with Technetium^{m99} and I-131 was normal.

Prior to surgery, needle aspiration confirmed the cystic nature of the mass. Two days later, the patient underwent neck exploration. The entire cystic mass was removed together with a small portion of the hyoid bone. The mass was not attached to the surrounding tissue or thyroid gland.

Examination of the gross specimen showed an ovoid-shaped cystic structure, pinkish tan in color with smooth glistening external surface, measuring 3.8 x 2.8 x 2.2 cm. On section of the structure, approximately 5 ml of dark yellowish liquid material was recovered. The inner surface of the cyst was smooth with evidence of solitary papillary excrescence in one area. The excrescence measured 0.2 cm at its greatest diameter. Histologic examination confirmed the cystic structure. The inner surface was lined with flattened to cuboidal epithelium mixed with simple columnar epithelium. One area of the inner aspect of the cyst showed papillary projections lined with cuboidal epithelium, arranged in a fibrovascular stalk (*Figures 1A* and *B*). The fibrous cyst wall showed cholesterol granulomas and a few thyroid follicles with colloid casts in the lumen. Scattered glandular tissue with papillary pattern was also seen intramurally. Some inflammatory infiltrates of lymphocytes were also seen in the supporting stroma. The histologic findings were consistent with the diagnosis of papillary carcinoma of thyroglossal duct cyst. Histologically the excised hyoid bone was normal.

The patient was discharged without medication and was advised to return six months later.

Thyroglossal duct cysts are common, but only rarely is such a mass found to be malignant. Reported here is a case in which histologic evaluation confirmed a diagnosis of papillary carcinoma. A review of the literature is presented.

Discussion

The thyroid gland develops from the foramen cecum during the embryonic life. The foramen cecum, which is located on the anterior floor of the pharyngeal gut, grows downward as the thyroglossal duct in front of the trachea and hyoid cartilage by the seventh week of gestation. By eight weeks, the thy-

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Figure 1A. Papillary carcinoma arising from the thyroglossal duct cyst with intramural neoplasm. (H & E 4x)



Figure 1B. Photomicrograph of a section of papillary excrescence through the wall of the thyroglossal duct cyst showing papillary carcinoma. (H & E 40x)

roglossal duct disappears. Persistence of the vestigial wall forms the thyroglossal cyst and sinus. These can be located on any part of the entire tract. The thyroglossal cyst is normally located on the midline near the hyoid bone, and is lined with cuboidal or squamous epithelium. The thyroglossal cyst usually presents as part of the differential diagnosis of a midline neck mass.[1] There seems to be an increased familial incidence[9] and also a tendency toward recurrence of this disease.[7] The incidence of carcinoma arising from the thyroglossal cyst is very low.[2, 3] There is controversy in regard to the origins of cancer in the cyst. Judd *et al.*[10] felt that cancer in the thyroglossal cyst represented an extension of occult thyroid cancer and advised total thyroid exploration and careful multiple biopsies. Others[11, 12] believe that the cancer arose de novo from the thyroglossal cyst. In their review of world literature, Joseph and Komorowski[5] found a total of 83 cases of carcinoma arising from thyroglossal cyst. They found that the tumor was more common in women (4:3). In their series, the most common presenting symptom was a swelling of the midline of the neck. Histologically, the cancer in a thyroglossal cyst is more often a papillary structure (74/83). Other malignant varieties in the series included adenomatous, follicular, and squamous cell types. In the majority of the reported cases, thyroid function tests — *i.e.*, serum thyroxin, RAI, uptake and scan — were normal.[6, 8, 9, 11, 12] Due to significant incidence of recurrence and neoplastic changes in the thyroglossal cyst, the Sistrunk technique[13] is used to remove the entire remnant of the thyroglossal tract and cyst. The material should be examined thoroughly histologically for any cancerous growth. Treatment of the disease varies from simple tumor excision[6, 8, 11] to near total thyroid excision, radioactive iodine-ablation of the thyroid gland, and thyroid suppression.[12] Longterm results of either of the modes of therapy have not been reported in the literature.

Acknowledgements

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Extracranial Carotid Disease

Noninvasive Evaluation

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STROKE is the third leading cause of death in the United States with 200,000 deaths occurring annually. Causes of stroke include intracranial carotid disease, extracranial carotid disease, and emboli. While intracranial pathology is most difficult to predict prior to a crippling stroke, such is not the case with extracranial carotid disease. Because approximately 40 per cent of all strokes are due to extracranial disease, early identification is important.[1, 2] Noninvasive procedures are important tools to identify extracranial arterial disease. They can also be used to measure its severity, follow its course, and to help determine the need for angiography and surgery.[3]

A noninvasive vascular laboratory was opened at St. Francis Regional Medical Center, Wichita in March 1980, and has the capability of evaluating the carotid system by a number of modalities including Echoflow, oculopneumoplethysmography, carotid audio analysis, and bidirectional doppler.

To evalute the effectiveness of one of these diagnostic tests — Echoflow — a prospective study was designed to compare the results of Echoflow with carotid arteriograms.

Materials

From July 1980 to July 1981, 1,317 patients were evaluated in the noninvasive laboratory with the Echoflow doppler imaging system. At a later date 212 of these patients had arteriograms, and this group is the basis of our study. The 212 patients consisted of 104 men and 108 women with an average age of 67 years.

Patients who were initially referred to the noninvasive vascular laboratory were either symptomatic with neck bruits, symptomatic without neck bruits, asymptomatic with neck bruits, or known to have atherosclerotic disease of other organ systems. Results were interpreted prior to and independent of the arteriogram. Arteriograms were ordered at the discretion of the primary physician. This was usually performed because of positive noninvasive studies or because of highly suggestive symptoms in the patient who had normal results from noninvasive studies.

Echoflow, a doppler imaging system which produces a color scan of arterial blood velocity in the carotid vessels, is a useful noninvasive procedure for detecting extracranal carotid disease.

Methods

The Echoflow doppler imaging system (Diagnostic Electronics Corporation) produces a color scan of arterial blood velocity. A primary application is examination of the carotid bifurcation. Here direct measurement can be made of carotid function in a noninvasive manner.[1, 4]

A picture of the carotid arteries is built up on a color display with each heartbeat, utilizing a position-sensing, hand-held probe. The three color, computer-processed image indicates red for normal velocity; slightly increased peak velocities are coded yellow; and markedly increased peak velocities are coded blue.[4] The blue color is representative of increased velocities due to more than 50 per cent narrowing of the vessel lumen which is a hemodynamically significant lesion.[1] This examination can be performed by a technician, and moderate and severe stenoses and occlusions are quickly identified. Although obstructive lesions are identified, ulcerative plaques are not identified by this procedure.

For the arteriograms, a transfemoral route with the Seldinger technique is preferred, and catheterization is achieved utilizing a French 5 Hanafee catheter to selectively visualize the common carotid arteries bilaterally. With the catheter located below the com-

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mon carotid artery bifurcations, biplane runs are made of the extracranial (magnification) and intracranial common carotid arteries. A French 7 or 8 straight catheter is then used for the aortic arch run with the patient in a right posterior oblique position. An anterior posterior run of the aortic arch and brachiocephalic vessels and a lateral run of the posterior fossa vessels are obtained. If a transfemoral route is unsuccessful, a transaxillary (either right or left) or a direct percutaneous cervical route is attempted.

Results

There were 212 patients (424 vessels) studied. Of these, six vessels were omitted due to inadequate arteriograms and nine vessels were omitted due to inability of Echoflow to scan the vessel — a problem encountered in patients with increased neck girth. This resulted in 409 vessels being included in the prospective study. Echoflow results correlated with the arteriogram findings in 355 of the 409 vessels for an accuracy rate of 87 per cent. In the 54 vessels that did not correlate there were 36 false positives (9% incidence) and 18 false negatives (4%).

Discussion

False positives were most often associated with a stenoic external carotid artery being imaged rather than the internal carotid, the Echoflow revealing a higher grade stenosis than the arteriogram, or technical difficulties separating the external and internal carotid arteries. Five of the false positives were interpretative errors. False negatives were associated with normal appearing velocity on the Echoflow when, in fact, the arteriogram showed 50-95 per cent narrowing. Five of the 18 false negatives were interpretative errors.

Patients particularly benefitted by the noninvasive Echoflow are those whose symptoms are not definite enough to initially warrant an arteriogram,[1] those with an asymptomatic carotid bruit,[3] and those with known atherosclerotic involvement of other organs. The highly symptomatic patients, such as those with transient ischemia attacks, will need an arteriogram regardless of the Echoflow findings as this test is designed to show obstructive lesions but will not detect ulcerative plaques.

Conclusion

In our evaluation of 212 patients who had both the Echoflow and arteriograms we found an 87 per cent accuracy of the Echoflow. Improvement due to experience should eliminate errors in interpretation and technique, and further increase the accuracy of the Echoflow. We therefore recommend this noninvasive procedure as a useful tool in evaluation of carotid artery disease.

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Exercise Training Program

(Continued from page 417)

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A Successful Partnership

I. Incidence, Awareness, and Control of Hypertension

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ALTHOUGH HYPERTENSION continues to be a major threat to the health of approximately 250,000 Kansans, the nature of the problem as it impacts health care providers is changing. The data collected through the University of Kansas School of Medicine-Wichita (UKSM-W) reflects a nationwide trend indicating an increase in individual awareness of hypertension as a personal health problem. However, there has also been an increase in the incidence of aware, but uncontrolled hypertension. The purpose of this article is not only to document these findings in Sedgwick County, but also to describe the screening outcomes of clients referred for further evaluation or treatment by a physician. Followup done on clients with elevated readings referred to their physician by the UKSM-W program indicate that the majority required initiation of, or a modification in their treatment plan from the physician, therefore indicating a successful partnership between community physicians and the UKSM-W hypertension program.

History

UKSM-Wichita, in partnership with the Medical Society of Sedgwick County, responded in 1973 to the national challenge to detect and counsel individuals found to have high blood pressure by founding the Hypertension Screening, Awareness, and Education Program (HSAEP) for Wichita-Sedgwick County citizens. Through the administration of the Division of Health Care Outreach, the goal of the program was, and continues to be, providing hypertension screening services to those individuals who might not seek assistance from private sources. It is to this task that emphasis has been placed on assisting minority populations, older citizens, and industrial workers. While target populations have changed, the numbers screened and counseled annually have risen from the initial 1200 adults in 1973 to 20,359 in the 1980 program. The total number screened during the seven year period is more than 108,000 individuals.

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Uncontrolled hypertension is a major health threat. The Division of Health Care Outreach at UKSM-Wichita has initiated a program to identify those who are unaware of their condition and to motivate all hypertensives to adhere to appropriate regimens for control.

Methodology

Protocols were established in 1973 to incorporate American Heart Association guidelines for detection and followup of hypertensive individuals. *Table 1* depicts the multitude of factors considered during screening clinics, as defined by the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, and adopted by the UKSM-W program in 1980.

While factors such as emotional status, environmental/occupational variables, physical stature, hereditary/genetic factors, and ethnic/cultural influences have been linked to hypertension, the evaluation of these complex and variable interactions are recognized to be in the domain of the physician. For the purpose of this community screening effort, several consistently elevated blood pressure readings regardless of the variables, serve as the basis for referral to a physician.

When an individual arrives at a screening site for blood pressure assessment, s/he remains at rest for at least five minutes prior to having blood pressure taken. Two blood pressure readings are obtained following the techniques recommended by the American Heart Association. The palpatory method is utilized for the initial assessment; the auscultatory method for the second reading; hence a double check of the value is noted. If an elevation is recorded, the individual is requested to return for a second screening within a month. This procedure is altered if the reading is $\geq$ 160/115; in this case immediate physician contact is encouraged.

TABLE I
ADULT BLOOD PRESSURE MEASUREMENT

I. Normal Screening Values
≤ 50 years = < 140/90
≥ 51 years = < 160/95

II. Referral Guidelines
Systolic Blood Pressure:
All adults ≥ 160 confirm promptly
Adults ≤ 35 yrs. of age & ≥ 150 mmHg confirm

Diastolic Blood Pressure:
All adults:
≥ 115 immediate physician referral
≥ 95 confirm within a month
≥ 90-95 confirm within 3 months

III. Classification
Stratum Diagnostic Reading
I. (Mild) 90-104 mmHg
II. (Moderate) 105-114 mmHg
III. (Severe) ≥ 115 mmHg

IV. Isolated Hypertension
Systolic ≥ 160 mmHg
Diastolic ≤ 90 mmHg
Borderline: 140/90-160/95

V. Elevated Blood Pressure
Two separate readings with
values ≥ the recommended value

Source: *The 1980 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure.*

Following the initial screening, a postcard is sent to each individual with an elevated blood pressure, notifying him/her of the need for a repeat blood pressure measurement and upcoming secondary screening sites. Upon the completion of this secondary screening, if the elevation persists, a referral is made to the individual's physician. If a private physician has not been obtained, the individual is encouraged to contact the Medical Society of Sedgwick County Referral Service for assistance. If an individual does not return for a repeat blood pressure measure within a month, telephone contact is initiated to encourage followup and attendance at a secondary screening clinic. The HSAEP does not diagnose or treat presenting problems.

While the program staff encourages self-responsibility for health and provides each participant in the program with a record of this individual blood pressure reading, the communication process is not completed until a record of the reading is also sent to the individual's physician. Many community physicians have come to appreciate the accuracy of the readings of the HSAEP staff and will record these readings in the patient's chart. Other physicians may use the information as cause to initiate contact with the individual for therapy modification.

Three months after the initial screening, contact is made by an HSAEP staff nurse with those individuals who had elevated readings on the secondary screening and were subsequently referred for physician care. The purpose of this contact is primarily to encourage physician contact, but also to provide any hypertension education that would assist in compliance with the prescribed regimen. The client is also contacted at a six-month interval for the same purpose. *Figure 1* depicts the screening follow-up protocol followed by the HSAEP.

Results

Nationally, an estimated 35 million individuals have been diagnosed as having hypertension. While this figure is staggering, it is also estimated that another 25 million could be affected, yet be unaware of their hypertensive state. With the emphasis recently placed on hypertension detection and follow-up, the number of undetected hypertensive individuals seems to be declining. In its place is an alarming trend for diagnosed hypertensive individuals to ignore treatment modalities; *e.g.*, to either refuse to see a physician or to fail to take prescribed medication. The reasons for non-compliance are as varied as the populations screened: lack of symptoms, increased symptoms with medication usage, side effects with medication administration, cost of treatment modalities, and denial of disease process. Data collected while providing the hypertension screening service to Sedgwick County residents reflect this trend. Although the data were not collected through a research model, the overall results are of interest, given the utilization of consistent blood pressure measurement technique utilized and the large number of individuals involved.

During the time period 1973-1975, a total of 47,310 individuals were screened, with 14.8 per cent (6,672) of participants having an elevated blood pressure. Of this total, 62.9 per cent had a previous

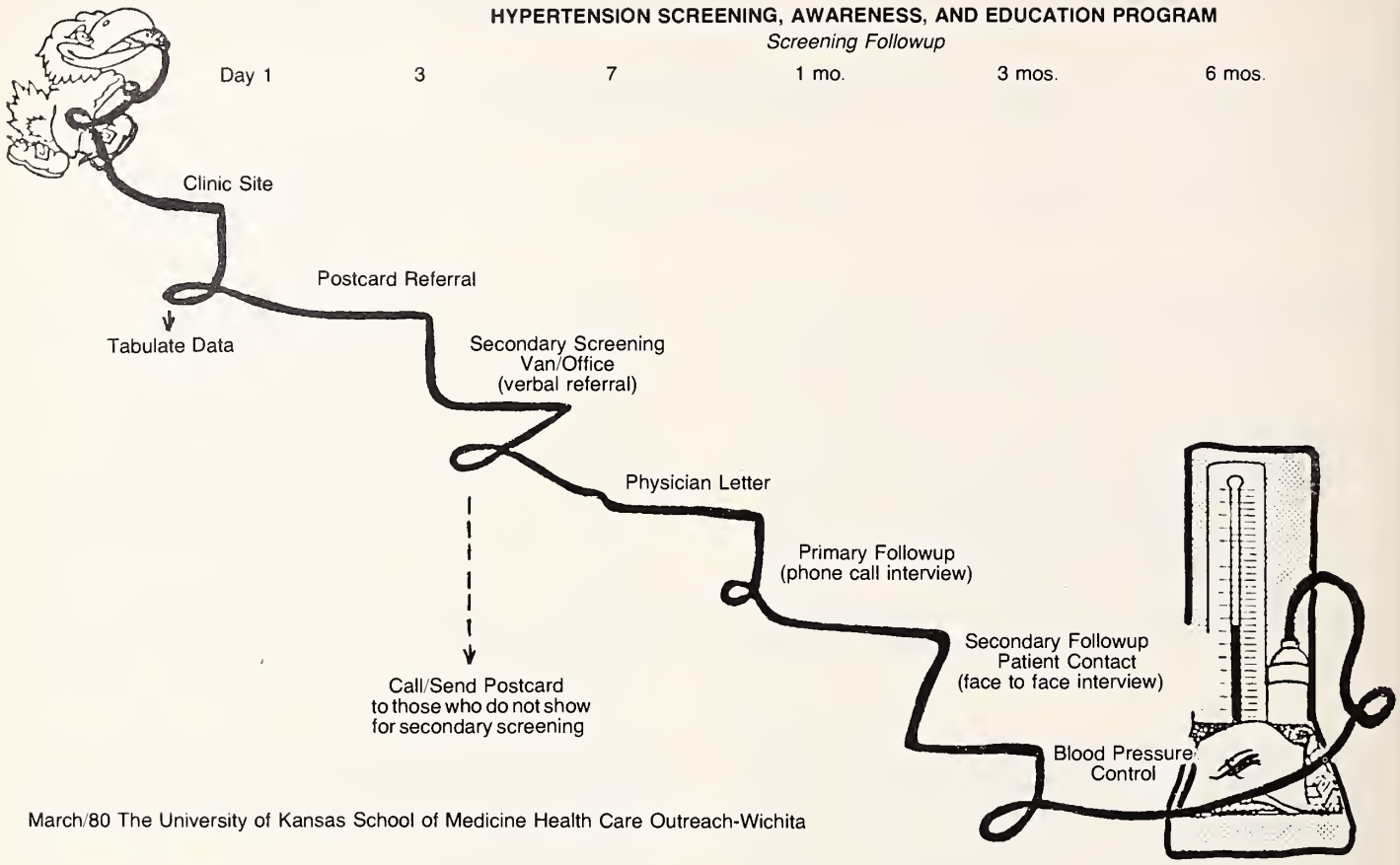


Figure 1. Screening followup protocol utilized by the HSAEP.

diagnosis of hypertension, and 37.1 percent were undiagnosed. Of those with an elevated reading, 1.6 per cent were on medication, 1.4 per cent had previously been on medication, and 97.0 per cent were not on medication. The incidence of hypertension, as noted by race, was as follows: 13.2 per cent (5,648) Caucasian; 22.6 per cent (1,103) Black; and 14.3 per cent (89) Hispanic.

Cumulative data for the time period 1973-1977 indicates that the program had screened 68,616 individuals with 13.2 per cent (9,058) having elevated blood pressures. Those diagnosed as hypertensive totaled 63.7 per cent, and undiagnosed was 36.3 per cent. Of those with elevated readings, 1.2 per cent were presently on hypertension medication; 1.3 per cent had previously been on hypertension medication; and 97.5 per cent were not on medication. The incidence of hypertension, as noted by race was: 12.7 per cent (7,390) Caucasian; 24 per cent (6,270) Black; and 12.9 per cent (804) Hispanic.

Beginning in 1977 the project was funded under alternative sources, and the data tabulation methods were subsequently modified and abbreviated. However, a project report issued in 1978, with a total of 71,443 individuals (cumulative time period 1973-1977) screened, noted an incidence of 16.3 per cent (11,050) with elevated blood pressure readings.

The first year for annual, non-cumulative, data tabulation was fiscal year 1979 (July 1978-June 1979). During that year a total of 8,347 individuals were screened; 845 individuals (10.2%) had elevated blood pressures. *Table II* displays the breakdown of screening data collected. A similar tabulation for fiscal year 1980 *(Table III)* documents an incidence of 12.6 per cent (1,057) with elevated blood pressure readings, of the total 8,401 screened.

During the fiscal year 1981, a total of 20,359 individuals were screened for hypertension in Sedgwick County, with 13.5 per cent noting an elevated blood pressure. The current program emphasis has been to provide screening services to high-risk and minority groups. During this time period, 71 per cent of the individuals screened were members of minority populations (Blacks, Hispanics, Native Americans, Indo-Chinese). A breakdown of incidence of high blood pressure by race is as follows: 9.9 per cent (n = 1,110 of total 11,125 screened) Caucasian; 19.6 per cent (n = 1,401 of total 7,147

TABLE II
HYPERTENSION SCREENING DATA/ Sedgwick County
July 1978 — June 1979
N = 8,347

	Elevated n = 845	*Non-Elevated n = 7,502*
No history	273 (32.3%)	5071 (66.9%)
History	273 (28.1%)	957 (12.7%)
On medication	335 (39.6%)	1528 (20.4%)
Column totals	845 (10.1%)	7502 (89.9%)

TABLE III
HYPERTENSION SCREENING DATA/Sedgwick County
July 1979 — June 1980
N = 8,401

	Elevated N = 1057	*Non-Elevated N = 7344*
No history	463 (43.8%)	5171 (70.4%)
History	272 (25.7%)	844 (11.5%)
On medication	322 (30.5%)	1329 (8.1%)
Column totals	1057 (12.6%)	7344 (87.4%)

screened) Black; 13.8 per cent (n = 166 of total 1,933 screened) Hispanic; and 6.17 per cent (n = 38 of total 626 screened) Indo-Chinese; and 13.2 per cent (n = 70 of total 531 screened) Native Americans.

As noted in the section on methodology, after confirmation of the elevated reading during a secondary screening, clients are referred for physician followup. After a three month time lapse, the UKSM-W staff contacts the client to assess the outcome of this referral or to provide further encouragement for the client to seek this physician contact. *Table IV* illustrates the form of compliance noted by 581 Sedgwick County clients with confirmed elevated readings in 1980, to HSAEP's referral for physician followup.

It should be noted that of those individuals with an elevated reading, 70.3 per cent made contact with their physicians. Of the 409 individuals making this physician contact, only 27.6 per cent (113) did not report a treatment modification in their prescribed plan.

Discussion

Reporting requirements have fluctuated according to changes in program philosophy and funding sources; therefore, only broad conclusions may be drawn. Foremost, however, may be the conclusion that the hypertensive population in Sedgwick County appears to be representative of reports presented in national studies, particularly as discussed in the recently published "Five Year Findings of the Hypertension Detection and Follow-up Program," *Journal of the American Medical Association,* (December 1979).

Interpretation of 1980-81 data documents the efficacy of the UKSM-W program in that 73.4 per cent of the individuals referred to their physicians required treatment or modification of their current regimen. The incidence of aware but uncontrolled hypertension is now considered a major problem since an increasing number of individuals fail to seek medical assistance or continue their treatment as prescribed by their physicians.

Given the continued need for community assistance, however, in detecting undetected hypertension, the Division of Health Care Outreach-Wichita, Hypertension Screening, Awareness, and Education Program will continue to seek to assist physicians in Southcentral Kansas in providing basic awareness of hypertensive problems through free screening clinics. It is believed that individuals can become more informed about their specific needs and can become more accountable for followup activities. Individuals can be referred to the UKSM-W for free blood pressure measurements; this not only allows more time for the physician to assist individuals who actively need his/her service, but provides a means to decrease health maintenance costs.

To assist in the need for control of detected hypertension, educational materials for both patient and professional groups can be obtained from HSAEP

for interested individuals or groups. One such program of the HSAEP, "It's Up to Me to Keep My Blood Pressure Down," provides both professional and patient education. Additionally, instructional software is also available for teaching staff or reaffirming personal skills. This program is described in the following section.

The need not only to assist newly identified hypertensive individuals in their understanding of high blood pressure, but also to assist the noncompliant client to comply with recommended treatment modalities, are ongoing goals of HSAEP. Assistance to minority populations, the elderly, and industrial workers in the detection and followup of hypertension will remain a high priority. Change to meet the demands of the Sedgwick County population will be ongoing, but the goal of HSAEP of the Division of Health Care Outreach-Wichita is to assist the community in maintaining positive health.

TABLE IV
TYPES OF COMPLIANCE REPORTED BY SCREENING CLIENTS WITH ELEVATED READINGS

	#	%
Physician seen/following a medication regimen	97	17
Physician seen/placed on an alternative treatment plan (diet, exercise, etc.)	18	3
Physician seen/treatment was revised	161	28
Physician seen/did not follow through with treatment plan (side effects, financial, other)	18	3
Physician seen/was not started on medication	113	19
Physician seen/directly admitted to hospital	2	.3
Has not followed through with seeing medical provider	113	19
All other individuals (unable to locate, etc.)	59	10.7
	N = 581	

II. A New Initiative for Hypertension Control

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While proven methods for controlling hypertension are available, it has been estimated that adequate hypertension control exists in only 29 per cent of the population.[1] Much success has been attributed to hypertension screening and detection efforts by the public and private sector in the identification of undetected hypertensives. However, it appears that the most difficult problem lies in insuring long-term control of hypertension through patient compliance to a prescribed regime. The prevalence of this problem is reflected in the previous section.

Because achieving this adherence to therapy is a common source of frustration to the physician, this article will describe a successful program sponsored by UKSM-W Division of Health Care Outreach which, in cooperation with community physicians, has achieved notable control of hypertension in participants.

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The program was initially funded through 317a PHS monies of the U. S. Department of Health and Human Services which were awarded to the Kansas Department of Health and Environment, and subsequently subcontracted to the UKSM-W, Division of Health Care Outreach-Wichita.

Review of the Literature

The medical literature documents the relative success, or lack of success, that various therapeutic modalities have had in achieving patient compliance in hypertension control (home blood pressure recording, group discussion, monitoring by pharmacist, home visits by a nurse, instruction pamphlet, lectures, drug blood level feedback, special medication packaging, contingency contracting, and others). However, Sackett and Haynes note that none of these measures is sucessful individually and positive results, in terms of client blood pressure reduction, can only be demonstrated in situations where a variety of modalities are utilized.[2] Most importantly the literature supports variations and combinations of two intervention techniques as having the greatest long-term impact on this chronic disease: continued patient education and the mod-

ification of the health provider's interactional process with the hypertensive patient.[3]

Education of the hypertensive patient has traditionally been effected through provision of factual health information through the use of printed material and patient tutorials. It is well recognized, however, that achieving and maintaining blood pressure control requires attitudinal, behavioral, and occasionally psychomotor skill development. Knowledge acquisition alone without change in behavior is not sufficient.

Learning is a process of behavioral change in the learner requiring more than factual knowledge. It may be provided through guidance, counseling, and support from the instructor (health care provider) with active involvement of the learner. These patient directed actions provide emotional support and guidance to influence behavior changes and affective learning.

Several studies demonstrate the effectiveness of this approach to hypertension patient learning. Schulman has reported that when provider-patient interaction patterns change, the patient becomes more involved in the treatment of the disease. His terminology for this process is titled "Activated Patient Orientation," and is positively correlated with treatment compliance.[4] The use of these techniques in the education of hypertensives increases the probability of therapeutic compliance. Solomon describes how implementation of these philosophical tenets in his medical practice has increased compliance with hypertensive regimens.[5]

The time constraints the average practitioner faces in developing and implementing programs for hypertension compliance led to development of a strategy to increase hypertension control in Sedgwick County. Of significance is evidence that indicates that, due to typical personality characteristics of the hypertensive, behavioral change could be increased through providing this educational process in a small-group setting. The small group with an intentional focus has been very effective in achieving control in other disorders (Alcoholics Anonymous, Weight Watchers).

Methodology

The program is designated as "It's Up to Me to Keep My Blood Pressure Down." During the first program year, the effects on blood pressure control of group education and group process skills were examined in 96 individuals with uncontrolled essential hypertension. Factors such as systolic and diastolic blood pressure reductions, client attitude changes, level of hypertension knowledge, and self-reported compliance to medication regimens were recorded for the group members.

Since the techniques utilized in the group education sessions have all been examined in other studies and the effects of patient education have also been documented, no attempt was made to prove or disprove these theories. The major focus of the project was to design, implement, and evaluate a group education program that could be presented by health professionals in the state of Kansas. The data presented represent a collective study of the effects of this group education program as presented by nurse educators in both urban and rural settings in Kansas.

The 96 individuals with diagnosed essential hypertension were studied during participation in a series of six sequential "class" sessions. Twelve groups of these "classes," with five to ten participants each, were conducted in Sedgwick, Butler, and Greenwood counties in Kansas. These multiple sites were utilized to represent persons residing in low, medium, and high population density areas in the development and implementation of the program.

Group members ranged from having been diagnosed as being hypertensive from one month to 361 months prior to the start of the class. Mean age was 58 years; 21 were male and 76 female. Eighty-nine reported their race as being Caucasian, seven were Black, and one Hispanic. The personal income levels of the group were clustered in the $6,000 to $15,000 range with 40 members of the groups reporting income within these limits. Thirty-eight reported completing high school, 32 had completed college, 12 had completed grade school education, eight members reported completing other forms of education, and seven members did not specify their educational attainment levels.

The group education curriculum consisted of six didactic programs in which cognitive information was presented in the form of a tape-slide sequence. Reinforcement of the information presented in the tape-slide sequence was provided to the nurse educator who discussed the factual material presented with the group. Practical methods of lifestyle adjustments to comply with the information presented are then discussed by the group members. Support and encouragement from the small group members provide a method by which individuals in the classes receive positive ongoing reinforcement to maintain these lifestyle changes.

The six educational components which comprise the "It's Up to Me" program are: (1) general knowledge about hypertension; (2) hypertension medications; (3) the effects of stress on blood pressure; (4)

TABLE I
MEAN SYSTOLIC BLOOD PRESSURE AND RELATED STATISICS BY WEEK OF GROUP PARTICIPANTS

	N	*Mean Systolic*	*S.D.*	*Minimum Value*	*Maximum Value*	*Standard Error of Mean*
Preclass	90	143.5	19.4	110	210	2.0
Week 1	91	138.0	14.1	106	184	1.5
2	92	133.0	13.6	98	162	1.4
3	91	133.7	12.1	106	172	1.3
4	90	130.7	11.3	104	162	1.2
5	91	131.7	12.5	110	168	1.2
6	89	129.9	11.0	110	166	1.2

nutrition issues and diet modifications; (5) lifestyle changes; and (6) personal decision making.

The behavioral nature of these group activities provide a method for the nurse educator to interact with each client as more than a giver of knowledge. The nurse educator facilitates group activity to provide a positive supporting environment to encourage lifestyle change within the individual and group as a whole. Participation by the client augments the group support system as each explores, within the group, the facets of hypertension, the control of this chronic disease, and the necessary lifestyle changes that this condition mandates.

Results

At each class session, the nurse educator determined the blood pressure measurements of each class participant using a mercury sphygmomanometer and standardized measurement techniques. Recognizing that the measurement of blood pressure varies with uncontrollable factors, a mean systolic and mean diastolic blood pressure were calculated for each session. The results of these measurements for systolic blood pressures are presented in *Table I* and for mean diastolic blood pressures in *Table II*.

In order to estimate the chance occurrence of the data presented in *Tables I* and *II*, a one tailed t-test was utilized. The paired one tailed t-test was used to analyze pre-class and post-class blood pressure data. Systolic pressures are displayed in *Table III* and diastolic blood pressures are shown in *Table IV*.

In order to assess the individual attitude change, each member completed during the first session and again during the last session, the multi-dimensional Health Locus of Control Test developed by Wallston, Wallston, and DeWellis.[6] This instrument is composed of three scales that measure health beliefs in relationship to internal influences (IMHLC), those that are externally influenced (PMHLC), and the belief that health is a matter of chance. Pretest and posttest difference given showed that statistically significant differences existed for the PMHLC and IMHLC scales, as displayed in *Table V*.

Cognitive knowledge about hypertension was measured using the Hypertension Education Level Test (HELT) developed by Wyka *et al.*[7] Pre- and post-class data analysis revealed statistically significant difference between the post-class and pre-class data. These results are displayed in *Table VI*.

TABLE II
MEAN DIASTOLIC BLOOD PRESSURE AND RELATED STATISICS BY WEEK OF GROUP PARTICIPATION

	N	*Mean Systolic*	*S.D.*	*Minimum Value*	*Maximum Value*	*Standard Error of Mean*
Preclass	90	84.4	10.4	60	110	1.1
Week 1	91	81.7	11.2	54	110	1.2
2	92	80.1	10.6	60	109	1.1
3	91	79.5	10.0	60	120	1.1
4	90	78.2	8.0	60	96	.8
5	91	77.7	9.0	60	100	.9
6	89	77.9	9.9	60	110	1.0

TABLE III
T-TEST ON SELF-PAIRED CLASS PARTICIPANTS FOR PRE-CLASS AND POST-CLASS SYSTOLIC BLOOD PRESSURES

$H_o: M_d = 0$
$H_a: M_p > 0$

$t(89) = 6.20$
$p < .01$
$M_{pr} = 143.52$ $s = 2.04$
$M_{pt} = 129.93$ $s = 1.17$

TABLE V
RESULTS ON ONE-TAILED T-TESTS ON PRE AND POST-CLASS IMHLC AND PMHLC DATA

$H_o: M_d = 0$
$H_a: M_p > 0$

IMHLC Pretest $M = 16.42$ $s = .31$
Posttest $M = 17.43$ $s = .29$
$t(89) = 2.91$ $p < .01$

PMHLC Pretest $= 13.88$ $s = .32$
Posttest $= 14.86$ $s = .31$
$t(89) = 2.53$ $p < .01$

Summary

Through the use of small group education techniques, hypertensives are taught about their disease, the nutritional and lifestyle changes that this disease may require, and positive health enhancing attitudes and behavioral changes are reinforced. Reductions in blood pressure and changes in hypertensive-related attitudes and knowledge were observed among class members in the group as a whole. While the evaluation methodology employed does not present a research design, the data should not be totally discounted. Despite the limitations of social research and funding sources, it appears that group education projects such as the "It's Up to Me" program may have impacted the problem of the aware but uncontrolled hypertensive. Program materials and training sessions are available now through the University of Kansas School of Medicine-Wichita. For more information contact RoxAnn Banks Dicker, R.N., M.N., UKSM-W, 1001 No. Minneapolis, Wichita KS 67214.

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TABLE IV
T-TEST ON SELF-PAIRED CLASS PARTICIPANTS FOR PRE-CLASS AND POST-CLASS DIASTOLIC READINGS

$H_o: M_d = 0$
$H_a: M_p > 0$

$t(89) = 5.99$ $p > .01$
$M_{pr} = 84.42$ $s = 1.09$
$M_{pt} = 77.89$ $s = 1.04$

TABLE VI
RESULTS OF ONE-TAILED T-TEST ON PRE-CLASS AND POST-CLASS HELT DATA

$H_o: M_d = 0$
$H_a: M_p > 0$

$t(89) = 7.60$ $p < .01$
$M_{pr} = 72.1$ $s = 1.8$
$M_{pt} = 82.3$ $s = 1.7$

Benign Liver Tumor

Report of a Case in a Newborn and Review of the Literature

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JOE J. LIN, M.D.,‡ *Wichita*

THE MAJORITY of pediatric hepatic masses are metastatic — originating from neuroblastoma, Wilms' tumor, rhabdomyosarcoma, or Hodgkin's lymphoma. Primary liver tumors comprise 15 per cent of pediatric abdominal tumors, and two-thirds of these are malignant.[1] These malignant liver tumors are most commonly hepatoblastoma or hepatocellular carcinoma, and account for 2 per cent of malignant childhood lesions.[2] Benign liver tumors are thus relatively rare. A recent case of a newborn with a hepatic mass treated at St. Francis Hospital, Wichita, stimulated this case report and a review of the literature.

Case Report

A newborn female was noted at birth to have a right upper quadrant mass, anemia, and thrombocytopenia. She appeared asymptomatic. The mass was palpable four fingers below the right costal margin and was felt to be nodular, firm, and nontender. The liver was palpable to the left of the midline. CT scan confirmed a right lobe liver lesion which enhanced with contrast; areas of decreased density throughout were felt to represent necrosis or cystic degeneration. It was entirely separate from the right kidney and retroperitoneal structures — all normal *(Figure 1)*. Laboratory data included: Hemoglobin, 6.9 gm/100 ml; hematocrit, 26.9%; white blood count, 21,700 with 2 bands and 60 segs; bilirubin, 2.8 mg/100 ml; direct, 0.4 mg/100 ml; alkaline phosphatase, 138 units; lactate dehydrogenase, 297 units; serum glutamic oxalo-acetic transaminase (SGOT), 31 units; alphafetoprotein, negative; urine, vanillylmandelic acid (VMA), negative.

At age 11 days the patient underwent exploratory laparotomy. A 4.5 x 6.0 x 6.5 cm mass was found in the anatomic right lobe of the liver. Right anatomic hepatic lobectomy was performed uneventfully. Frozen and permanent section diagnosis of hepatic mesenchymal hamartoma was made. Postoperative complications were limited to right pneumothorax responding to closed tube thoracostomy and right middle lobe pneumonia which resolved with antibiotics. Mild congestive heart failure responded to diuretics. The patient was dismissed on the 18th postoperative day, and no sequelae have been noted during the subsequent five months.

Among pediatric patients, the majority of hepatic masses are metastatic or malignant primary tumors. Reported here is a case of an infant with a rare benign hepatic mass.

Discussion

A 1975 report by the Surgical Section of the American Academy of Pediatrics[3] reviewed the types of benign liver tumors *(Table 1)*. It is noted from the graph that approximately one-third of liver tumors are benign; the most common lesion is hemangioma followed by hamartoma and hemangioendothelioma.

Most of these present with an asymptomatic mass or enlarging abdominal girth; abdominal pain, weight loss, and irritability are uncommon and are signs of advanced malignancy. The bilirubin is usually normal with alkaline phosphatase and SGOT normal or slightly elevated. Alphafetoprotein (an alphaglobulin present in fetal serum and normal infants during the first few days of life) is not present. It is found in two-thirds of malignant hepatic epithelial neoplasms. Anemia can be caused by bleeding into the tumor.

A 24-hour urine collection for VMA and homovanillic acid will aid in differentiating neuroblastoma; intravenous pyelogram is also used in this regard.

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Figure 1. CT Scan.



Figure 2. Gross specimen.

CT scan and ultrasonography will also aid in differentiating hepatic from retroperitoneal lesions.

The tumor discovered in our patient led to a review of the English language literature. Prior to this case there have been 29 reported cases of mesenchymal hamartoma.[4] This is the second reported case diagnosed in a newborn, both of whom had resection during the second week of life. Twenty-four of the cases were discovered prior to age 2 years; 12 within the first year of life, and four before age 3 months. The eldest reported case was discovered at age 8 years.[4] This lesion was first reported in 1903 by Maresch;[5] Edmondson in 1956 reviewed the literature and proposed the currently used name.[6]

The specimen, gross and microscopic, in our case is seen in *Figures 2* and *3*. It measured 4.5 x 6.0 x 6.5 cm; the largest reported mesenchymal hamartoma measured 30 x 30 x 20 cm.[7] A hamartoma is composed of tissues indigenous to the site (mesoderm and endoderm in the liver). It is felt to be due to faulty embryonal development. An admixture of loose connective tissue resembling the primitive mesodermal layer and ductal structures is present. Liver cells are seen mainly in the periphery of the tumor and are distorted. Mitoses and invasion of adjacent liver is absent. Fluid can accumulate in quantities sufficient to create micro- and macroscopic acellular spaces suggestive of lymphangiomatous channels with endothelial lining. These

(Continued on page 445)

TABLE I
PATHOLOGY

Benign	
Hemangioma	38
Hamartoma	37
Hemangioendothelioma	16
Adenoma	7
Focal hyperplasia	4
Cholangiohepatoma	1
Eipthelioid epithelioma	1
Benign cyst	10
Hydatid cyst	6
Eosinophilic granuloma	1
Lymphangioma	2
	123

Types of benign liver tumors.
Surgical Section of the American Academy of Pediatrics 1975.

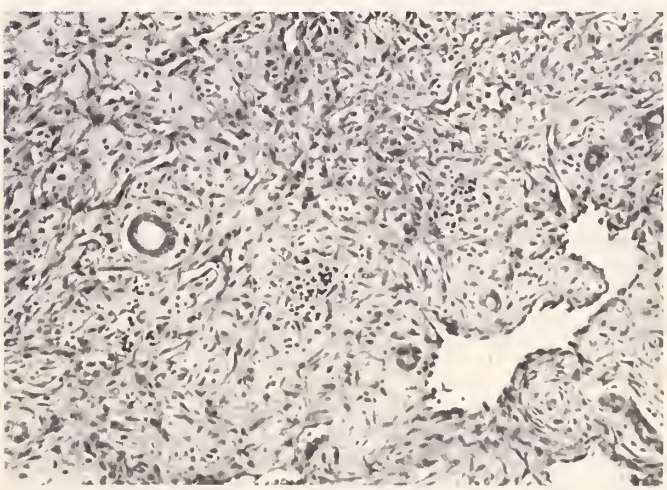


Figure 3. Microscopic specimen.



Justice For All

Abolition of the Defense of Insanity

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THE DEFENSE of insanity has been a part of Anglo-American jurisprudence almost from its inception. Initially, the laws were very simple and straightforward. The defense of insanity arose only when the accused was clearly insane, and was recognized as such by both the prosecution and the defense. It was an attempt to be humanistic in a situation where both sides were big loosers. The accused was inflicted and stigmatized with "insanity," which at that time meant a lifetime of such degradation and scorn that many often opted for death. It was a tragedy for all, but no reparation was possible.

The nineteenth century ushered in a new era in the insanity defense with the M'Naghten trial being the most celebrated case. This began an era of confusion. The insanity defense was so abased, manipulated, and distorted that today it represents injustice, not humanity. With the current mental health system, the imprecision of psychiatry and legal precedents, true justice cannot be served in our country until the defense of insanity is abolished.

Historical Background

Since Roman times merciful judges and peers have exculpated wrongdoers of their crimes if it was determined that the criminal acted without a "free will" in committing the offense.

Present Anglo-American laws regarding the mentally ill are derived from Roman law, Judeo-Christian legacy, and the moral philosophies of Plato and Aristotle. English common law is an amalgam of pre-Norman conquest sectional law and Canon law. Canon law insisted that crime must involve moral guilt, or guilty intent, while crimes in common law have two essential elements — the act and the intent.[1]

In the seventeenth century, Sir Matthew Hale wrote that "most persons that are felons . . . are under a degree of partial insanity. . . . It is very difficult to define the invisible line that divides perfect and partial insanity. . . ."[1] Hale advocated that only total insanity should bar punishment. Total insanity was redefined in 1724 when Justice Tracey told a jury that to be acquitted "a man must be totally deprived of his understanding and memory, so as not to know what he is doing, no more than an infant, a

The legal mediation of crime and mental illness has resulted in gross injustices to all involved. Abolition of the defense of insanity and revision of this segment of criminal jurisprudence is requisite to assure justice for all.

Editor's Note: Papers published in the Student Thesis series have been written by fourth year medical students at the University of Kansas School of Medicine. Those selected for publication have been recommended by the UKSM faculty.

brute, or a wild beast.''[1] Soon after, the emphasis shifted from knowing what was morally ''good and evil'' to knowing what was legally ''right and wrong.''

The ''wild beast'' test — *i.e.* the ''total insanity'' ruling — was mitigated in 1800 in the Hadfield case which marked a radical change in the attitude of the court. Previously, the defendant was determined to be legally responsible for his crime if he could be shown to have minutiae of reason. Subsequently, several standards were used to determine if the defendant was exculpably insane.[1]

State and federal courts presently use four tests of criminal responsibility to evaluate and convict or acquit an offender with possible mental illness. They are the M'Naghten rule, the M'Naghten rule with the irresistible-impulse test, the Durham or product test, and the American Law Institute's Model Penal Code (ALI test).[2]

M'Naghten Rule

The first major test for determining criminal responsibility was the M'Naghten test, formulated in the 1840s. It states that ''to establish a defense on the ground of insanity, it must be clearly proved that at the time of the committing of the act, the party accused was laboring under such a defective reason from disease of the mind as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know he was doing what was wrong.''[2] The popularity of the M'Naghten test is now waning; it is currently used in less than half of the states.

The M'Naghten rule attempted to answer the question of how much insanity must be present to declare a person legally insane.[3] In the process it raised even more questions. One no longer had to be totally insane to be acquited, as now it was recognized that varying degrees of ''insanity'' and ''knowing'' existed. Where was the line to be drawn? The M'Naghten defense relied heavily on the views of the psychiatrist to show whether enough ''knowing'' was present in the defendant to declare him legally responsible. This led the way to the establishment of the psychiatrist as the central figure and, oftentimes, chief decision maker in cases involving insanity.

Irresistible-Impulse Test

The M'Naghten test with the addition of the irresistible impulse formulation is used in 15 states to determine criminal responsibility in insanity cases. Under the irresistible-impulse test, an offender is exculpated if it is determined that he was forced to commit the crime because of an irresistible impulse over which he had no power to resist, even though he may have known right from wrong.[2]

Some found this test to be more humane than previous laws. The reasoning was that a person could not be rehabilitated if he had no power to control his actions; therefore he should be acquitted. The concept that nondeterrables should be exculpated would eventually complicate the issue of legal insanity as critics pointed out that prisons were filled with nondeterrable hardened criminals.[3]

Durham Test or Product

In the 1870s, the Supreme Court of New Hampshire added to the mounting body of laws regarding criminal insanity when it ruled that a person could not be held responsible for an act that was the ''product'' of mental disease. This ''product'' test gained wider acceptance in 1954 when the U. S. Court of Appeals acquitted Durham because his criminal act was determined to be a ''product of mental disease or mental defect.'' The Durham test encompassed not only the spontaneous irresisible act, but also the criminal act that was the result of much reflection over a period of time.[2]

The Durham test also established that the mental state of the offender at the time of the criminal act must be used in determining sanity. Since an examination of the defendant was not likely to have been performed at that time, expert testimony regarding the probable mental state of the defendant at the time of the offense, based on subsequent examination, was deemed acceptable. Finkel sums up the result. ''A psychiatrist testifying under M'Naghten rules must produce evidence regarding the defendant's mental state and responsibility at the time of the act; a psychiatrist testifying under Durham rules need only produce hunches embracing the time of the act. The quality of the evidence has indeed changed — it has diminished. . . .''[3] In addition to diminishing the quality of the evidence, the Durham case further established the psychiatrist as the central figure in insanity trials, with expert testimony, or ''hunches,'' becoming even more important.

Leifer contends that the Durham trial led to such a reliance on experts that psychiatrists could subsequently testify in court with few restrictions, using their own personal parlance and theories.[4]

Discussion

The defense of insanity needs to be abolished. It has progressed to, or rather deteriorated to, a point far from its original intent. The current system is

replete with incongruities, injustices, and failures. Many noted psychiatrists, lawyers, and scholars are supporting the abolition of the insanity defense. Some do so because of their interest in the judicial system, some because of their interest in those deemed "insane," and others because of their interest in justice in America. Some recent abolitionists include Chief Justice Weintraub, Dr. Karl Menninger, former Justice Abe Fortas, Lady Barbara Wootten, Professor H. L. A. Hart, Dr. Thomas Szasz, and Professor Norval Morris.[5]

As can be ascertained from the historical review presented, qualifications for the defense of insanity have changed markedly over the years. Before M'Naghten, courts had only to determine if a defendant were "totally" insane, or as a "wild beast" to be acquitted because of insanity. After M'Naghten, courts had to answer many vital questions to determine whether an offender could use the insanity defense. What degree or amount of insanity should constitute legal insanity? How much "knowing" or "capacity" is "substantial" or "adequate?" Was the lack of resistance to a criminal urge because of an inability (irresistible-impulse) to resist, or simply because of a failure to resist? How conscious versus unconscious was the act? And, recognizing that even an insane person can often make rational choices, was the lack of resistance to the criminal act because of the mental incapacity or was it secondary to calculated indisposition?

Courts continued to define legal insanity and criminal irresponsibility in vague terms. Someone had to determine if the defendant conformed to those definitions. Judges and jurors were reluctant to rely on their own best guesses in making such an important decision, so they called on the psychiarists. Mental health personnel, promoting psychiatry as a science, would give "expert testimony" such as a physicist or an engineer would explain a scientific phenomenon. Yet, because much of psychiatry is based on theories rather than on proven scientific principles, decisions regarding the mental state of the defendant were not "scientific" at all, but were opinions or "hunches" as Finkel prefers.[3] So in reality psychiatrists were no more capable of providing the answers than the jurors were. Answers by anyone are actually opinions, and opinions — whether by judge, jurors, or psychiatrists — cannot be regarded as absolute, factual, or scientific. An exception would be the defendant, who might know his exact capacity and intent, but who is unlikely to incriminate himself. Like all others, not even he could determine with certainty where he fits on the ladder of mental health, where one rung means the diference between guilt and innocence.

Leifer feels that psychiatrists knowingly guide and misguide juries. He contends that the judge and jurors rarely understand the technical jargon used by psychiatrists, so they must rely on the psychiatrists' conclusions or opinions as to whether or not the act was a result of the mental illness. As a result, the jury is more influenced by the psychiatrists than by the facts. In reality, it is the psychiatrists, not the jury, who makes the moral decision.[4]

Wertham alleges that jurists are often subtly prejudiced by the psychiatrist's testimony. Jurors may think that if they do not accept the expert testimony, they could be considered unsophisticated or ignorant. As a result, they may accept even hightly speculative claims.

So in reality, the decisions of the judges and jurors are being determined by psychiatrists, who are most likely bringing a humanistic and sympathetic philosophy, rather than scientific testimony, into the courtroom. Justice is being diluted as offenders are acquitted. The judges and jurors continue to shirk their responsibility for making a decision regarding the true guilt or innocence of an offender while relying on hunches, best guesses, and opinions of psychiatrists regarding the defendant's intent, capacity, will, consciousness, and knowledge.

Judges and jurors need to start assuming the responsibility that is theirs. Mental health experts need to stop pretending they have the answers. Also, the courts need to return to the questions that are at issue in all other criminal proceedings — *i.e.* whether the offense is right or wrong, and whether the accused is guilty or innocent of the charge.

Responsibility in people with mental illness is not an all or none phenomenon as once thought. Wertham stated that even the psychotic cannot completely explain the murder he commits by his insanity.[6] Varying degrees of knowing, intent, and capacity exist. Therefore, all offenses committed by all people with varying degrees of insanity should not be treated equally under the criminal law. That is what legally occurs with the defense of "innocent by reason of insanity." There are no legal levels to allow for the grays. All are treated alike.

We now have a justice system that cannot really determine where the dividing line should be in the continuum from mental health to mental collapse. Yet, we pretend that we can assign one person as guilty and the other as innocent. A system that is so arbitrary that the people on rungs five and seven of the ladder are deemed innocent (by reason of insan-

ity), but the person on rung six is deemed guilty does not deliver justice. Not only is the one on rung six ill served, but so are those on rungs five and seven who may be on opposite ends of the spectrum regarding intent, capacity, and knowledge.

The problem can be solved only by abolishing the defense of insanity. Insane people ought to be treated as all other people in a court of law. We need to cease giving them concessions that were originally adopted for "wild beasts." Two questions need to be addressed by the court: Was the act that was commited wrong, or against the law? If so, is the person charged guilty or innocent of that charge?

Mental incapacity should not be a defense; it is an extenuating circumstance. Finkel states "these 'mental conditions' are a mitigating, not an exculpating, factor. . . . Whether a man kills another with thoughful premeditation or sudden passion; or because he is an alcoholic — drunk and driving — or is mentally unbalanced does not, in my opinion, remove responsibility entirely."[3] In determining the verdict, only the same two conditions should apply in all cases — wrongdoing, and then guilt or innocence. This is justice that serves all.

As is the case in all other forms of criminal jurisprudence, the sentencing is the area where judge and jurors should have flexibility, the minimum and maximum allowable sentences for a given offense are set by the legislatures. This range allows juries and judges to take into account the norms of the period and locality, and any extenuating circumstances applicable to the defendant or to that case in deciding the sentence. If our laws were such that insanity was recognized as an extenuating circumstance involving an offender, sentencing legislation scaled for various types of offenses and circumstances could be enacted. Since intent to do serious bodily harm must be proved for a conviction of first and second degree murder, murder by someone deemed insane could be treated as manslaughter, or even as a newly legislated lesser offense. Where these offenders would serve their sentence could also be legislated according to the wants of the constituency. Whether mentally ill criminals receive treatment (hospitalization) or punishment (imprisonment) is a highly controversial issue. Another alternative would be a separate institution for the criminally insane, as was once common, with a staff including psychiatrists, psychologists, social workers, teachers, and other mental health team members.

With our present system which allows acquittals at all levels of mental incompetence, and for all offenses, no matter how grievous, injustice is served not only to all innocent people in our country, including the "innocently insane," but also to the criminally insane themselves. So much as been written about this aspect that further elaboration is in order.

The most controversial issue is the injustice that our present system imposes on those truly incompetent who have committed crimes. What happens to those offenders who are now acquitted by reason of insanity? Again confusion and chaos reign. Some people believe that if the mentally ill offender is deemed innocent, albeit by reason of insanity, he should be free of any further obligation to the state, as is the case with non-ill defendants. They think it is an incongruity to declare a person innocent, and then impose a sentence. They point out the similarity of incarceration in prison and incarceration in mental hospitals. Others feel that sentencing is just providing "treatment" for needy ill people, while keeping the dangerous ones off the street.

Some states require the jury to determine whether the offender is still insane at the time of the trial. If they determine the acquitted to be no longer insane, they must set him free. However, in other states no determination is made about current sanity, and the acquitted is subject to mandatory hospitalization until discharged as sane.[5]

The methods of discharging the offender from the mental institution are also controversial. Some states require a rehearing of the hospitalization order or a discharge proceeding; however in a number of states the mental institution itself, or its psychiatrists, may discharge a person without a court order.[5]

The whole process has incensed both liberals, who want more rights for the insane, and conservatives, who feel an epidemic of crime is upon our country and want safer streets.

Szasz, a prominent psychiatrist and author, favors abolition of the insanity defense and reorganization of our methods of dealing with the criminally insane. He contends that these people often have less stimultion, space, and personal freedom in hospitals than criminals have in prisons. He writes, "The existence of psychiatric institutions that function as prisons, and of judicial sentences that are, in effect, indeterminate sentences to such prisons, is the backdrop against which all discussion of criminal responsibility must take place. . . . For what does it matter whether or not the accused was, at the time of the offense, 'sane' and criminally responsible or 'insane' and criminally not responsible. . . . In the final analysis, the insanity plea and the insanity

verdict, together with the prison sentences called 'treatments' served in buildings called 'hospitals,' are all parts of the complex structure of institutional psychiatry which, as I have show, is slavery disguised as therapy. Those who value and wish to defend individual liberty can be satisfied with nothing less than the abolition of this crime against humanity.''[7]

Finkel concurs with Szasz, and points out that the average length of psychiatric hospitalization involving acquitted mental patients is longer than the average length of imprisonment of criminals. He also points out that the mentally ill offenders frequently have no idea when they might be discharged, and that often they never are.[3]

With the abolition of the defense of insanity, determinate sentences enacted by legislation could be imposed on each offender. The time could be served in an institution determined by the people of the jurisdiction, through legislation, to be just.

On the other side of the coin, injustice is served to those law abiding citizens who strive for a good life for themselves and their families only to be victimized because a ''lenient'' and ''humanistic'' judge or psychiatrist believed that partial mental incompetence in an offender automatically removed all of his reponsibility. Examples abound in which a defendant ''not guilty because of insanity'' is released from a mental institution only to maim, torture, rape, or murder again. Several years ago, *Newsweek* described a case in which the insanity defense was used, and within a year the acquitted had murdered again. The reaction to the insanity defense was summed up, ''The Vanda case outrages Chicagoans who think the insanity defense is little more than a ploy used by killers, abetted by crafty defense lawyers and soft hearted psychiatrists, to escape punishment.''[8] Justice would be served to Chicagoans, and to all Americans, by abolishing the defense of insanity and sentencing these offenders to spend a determinate period in a specified institution.

Perhaps the greatest injustice of our present system is imposed on the insane who never commit a murder, those who live by a strong sense of moral obligation and legal responsibility for their acts. Isaac Ray stated in 1838, ''That the insane mind is not entirely deprived of this power of moral discernment, but in many subjects is perfectly rational, and displays the exercise of a sound and well-balanced mind, is one of those facts now so well established, that to question it would only betray the height of ignorance and presumption.''[9]

Currently, few institutions exist specifically for the criminally insane. In our country today, state mental facilities are used to house those offenders determined to be insane. As such, non-offender patients in these hospitals are forced to co-exist with the insane offenders of all degrees of dangerousness. In practice, the truly innocent mentally ill often have fewer rights than those criminally insane.

With the insanity defense, all insane people are categorized as the same and all are forced to live in the same institutions. As a result, the general public, which has had little contact with the mentally ill, begins to think of them as all the same. Some even consider all mentally ill patients to be dangerous. When a person is discharged from a mental hospital to the community, there tends to be fear in the town. Citizens lock their doors and keep their children inside. The mentally ill offenders have stigmatized even those innocent, responsible mentally ill.

Others, who pride themselves with being ''sympathetic'' and ''humanistic'' to the needs of the mentally ill, also begin to put both the offenders and non-offenders in the same class. There is a tendency for them, while crusading for the rights of the mentally ill, to also begin crusading for more sympathy and leniency for all criminally insane, and then by extension, for all criminals. It has often been ''liberal'' and popular to espouse the theory that ''after all, all criminals must be at least partially insane.'' Reasoning like that blackens the reputation of the innocently insane as well as the criminally insane, and stigmatizes them. Wertham states that, ''By pretending that every murderer falls into the category of mental pathology, we deprive the genuinely sick of proper consideration, and we deprive the community of protection.''[6] Some criminals rape, murder, mutilate, and terrorize for their own selfish gratification. Just as these offenders should not be classifed as the same as those insane who commit the same acts, the insane who are truly innocent have a right to be separated physically and legally from those insane who rape, murder, mutilate, and terrorize.

There are many examples of injustice that could be eliminated or mitigated with the abolition of the insanity defense and with sentences that fit the crime while adjusting for mitigating factors.

Some criminals may pretend to be mentally ill to get an easy acquittal. Wertham states, ''A subject that has been left in considerable twilight in forensic psychiatry is that of simulation of major mental disease. . . . Often a fellow prisoner, especially one who has himself been under psychiatric observation in a hospital, tells a man in jail how to behave so as to be sent to a psychiatric ward and how to act when he gets there. Sometimes a successful simulator in-

stucts others even to the extent that he supplies the replies that should be given on intelligence and Rorschach tests. Over the years. . . . I have collected a large number of cases of simulation . . . in which followup studies of ten or more years prove the diagnosis . . . simulation of a psychosis in order to evade punishment is at present not rare. . . . A criminal facing severe punishment grasps at every straw."[6]

Wertham also states that often it is the defense lawyer who grabs for the insanity defense. "A violent crime is committed, and the lawyer feels he has no valid defense, therefore he pleads insanity. . . . The abuse of the insanity defense has led to what may be called the alibi of abnormality. Instability is not insanity, maladjustment not psychosis, a partial explanation not a total excuse."[6] The jury is asked to correctly call one gray "black" or guilty and another gray "white" or innocent. This is difficult when only the facts are presented, but impossible when lawyers, defendants, and expert witnesses skew the facts.

Conclusion

In conclusion, it is time that the defense of insanity be abolished. The current system was born under antiquated, disproven theories and has grown out of haphazard amendments. The result is an amalgam of confusing and unjust laws based on unsound principles. These vague laws have become easy prey to be manipulated and distorted by unscrupulous lawyers, mental health professionals, hardened criminals, and self-serving defendants. With the abolition of the defense of insanity and a reorganization of this area of criminal jurisprudence, these ambiguities would be eliminated and justice better served.

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Benign Liver Tumor

(Continued from page 439)

cysts are multiple and multilocular grossly, producing irregular surfaces with walls of variable thickness. The mesenchymal overgrowth can make the lesion partly or mostly solid so that the cysts are seen only on cut surface. The lesion is usually in the right lobe and is attached by a pedicle in approximately one-third of cases.[7]

Treatment of benign pediatric liver tumors is surgical; complete resection by enucleation, excision or formal lobection, if feasible, should be performed. Resection beyond the tumor into normal liver is unnecessary when this diagnosis can be established at the time of operation. Exploration and biopsy may be necessary before definite excision. A blind percutaneous biopsy is of little help as it is difficult to obtain a representative specimen.

Summary

The second reported case of mesenchymal hamartoma in a newborn has been presented. Benign liver tumors and their treatment have been reviewed.

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Current
COMMENT

H. B. LINDSLEY, M.D., *Editor*

Travelers' Diarrhea

Recent Advances in Prophylaxis and Treatment

ANNE P. MARSH, M.D., *and* MARK S. McPHEE, M.D., *Kansas City, Kansas*

More than one-quarter billion people travel from one country to another every year. American travelers to Mexico alone number 3 million annually. Of these, 25-50 per cent will experience an acute diarrheal illness during the trip. "Travelers' diarrhea" is defined for study purposes as a two to threefold increase in the usual number of stools/24 hours, most often accompanied by a change to liquid or watery consistency. Associated symptoms include cramping abdominal pain, fever, chills, and nausea or vomiting. The typical case, due to *E. coli,* is not associated with bloody or purulent diarrhea, and severe systemic toxicity is usually absent. A customary and even anticipated "travelers' companion" in many parts of the world, this form of diarrhea is regionally known by such colorful names as the "Aztec two-step," "Montezuma's revenge," "Hong Kong dog," "Delhi belly," "Casablanca crud" and, in travelers to the Soviet Union who acquire *Giardia,* the "Trotskys."

Typically, the traveler feels well for the first two or three days of the trip, experiencing abrupt onset of illness four to six days after arrival. Abdominal cramps are followed by watery diarrhea, which lasts an average of two to three days — just long enough to ruin most vacations or business trips. Travelers may occasionally experience several separate episodes of diarrhea during a single stay, but onset of illness may be delayed until after return home. Symptoms may sometimes persist for weeks.

Epidemiology

The most important determinant of attack rate in travelers' diarrhea is the destination of the trip. The incidence of diarrhea in American travelers to Mexico, Latin Ameria, Africa, and the Mediterranean, Middle East, and Far East is 25-50 per cent. Low risk areas include the British Isles, Scandinavia, northern and western Europe, and Canada. Most areas of Asia are intermediate in risk (15%). The traveler's country of origin is also of importance; individuals at risk tend to come from temperate countries with a low incidence of endemic infective diarrhea. For example, at a conference held in Tehran,[1] diarrhea occurred in 41 per cent of North Americans, South Africans, and western Europeans, compared with only 8 per cent of visitors from the Middle East, Southern Europe, and Brazil.

Surprisingly, the oft-quoted correlation between travelers' diarrhea and eating unwashed fruit or drinking tap water is rather poor, although there may be a slightly higher risk with consumption of fresh salads. Of apparent greater importance is the avoidance of certain "culprit" restaurants. Eating more than 50 per cent of meals at relatives' homes, rather than in local restaurants, is known to be associated with a much lower risk of diarrhea. Young people are more susceptible, as are those taking antimotility agents or cimetidine.

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Etiologic Organisms

Travelers' diarrhea is only rarely due to amebiasis. In fact, *Entamoeba histolytica* is such an uncommon cause of travelers' diarrhea that it has been called "the refuge of the diagnostically destitute."[2] Recent studies have shown that enterotoxigenic *E. coli* (ETEC) cause some 40-70 per cent of cases of travelers' diarrhea. This percentage is remarkably constant worldwide. Additional common pathogens include *Salmonella* and *Shigella* species, with a reported incidence of approximately 5 per cent each. *Giardia lamblia* is a relatively common pathogen worldwide (5% of total cases), but in areas of endemic giardiasis such as the Soviet Union, the attack rate may be as high as 50 per cent. Viruses, principally rotavirus, are probably responsible for less than 5 per cent of cases. Other uncommon pathogens include *Vibrio cholerae,* which produces cholera and related illnesses, and *Vibrio parahaemolyticus,* which classically is responsible for diarrhea after eating shellfish. Despite careful culture techniques, no specific etiologic organism can be found in 10-35 per cent of cases. The role of *Campylobacter fetus* has not yet been clearly defined in travelers' diarrhea, but it may be responsible for many of these "culture-negative" cases.

ETEC usually cause disease by colonizing the small bowel and elaborating an enterotoxin which results in massive intestinal fluid secretion and concomitant watery diarrhea. Characteristically, the illness is brief (1-2 days), relatively painless, and without fever or evidence of mucosal invasion. Rarely, *E. coli* may be enteroinvasive. This organism can produce severe diarrheal illnesses with enterocolitis indistinguishable from shigellosis except by culture. The ability of *E. coli* subspecies to secrete enterotoxin is not related to serotype, hence ETEC are indistinguishable on routine culture from other *E. coli,* except by laboratory identification of one of the two known enterotoxins.

Prophylaxis

The frequency of travelers' diarrhea has led to a widening search for effective prophylaxis. General dietary measures, such as avoidance of tap water and fresh fruits, have been shown in several studies to be of almost no benefit. Multiple antibiotics have been tried and have been shown either to be ineffective as prophylactic agents or to have unacceptable side effects. Most recently trimethoprim-sulfa (Bactrim) has been evaluated; some preliminary reports suggest earlier development of antibiotic resistance and higher worldwide incidence of already resistant strains. Since more than 90 per cent of the strains of *E. coli* detected in the original studies were sensitive to tetracycline, several recent trials have been performed using doxycycline as prophylaxis. Doxycycline was chosen from among the tetracycline family because of its long half-life, broad spectrum, and high achievable concentrations in the gut.

The first study, by Sack *et al.*,[3] was a randomized double-blind study of 39 Peace Corps volunteers in Kenya. The volunteers took either placebo or doxycycline (100 mg/day) for three weeks and then were observed for an additional two weeks. Forty-three per cent of those taking placebo, but only 6 per cent taking doxycycline developed travelers' diarrhea during the treatment period. However, anti-diarrheal protection for the doxycycline group persisted for just slightly more than one week after treatment was discontinued.

In a second study by Sack,[4] 50 Peace Corps volunteers in Morocco took either doxycycline or placebo for three weeks, and were observed for an additional seven weeks. Forty-six per cent taking placebo and 8 per cent taking doxycycline developed travelers' diarrhea during the treatment period. One week after discontinuing doxycycline, however, several episodes of diarrhea had developed; and by three weeks, the frequency of diarrhea was identical in both groups (54%). Sack also performed a third study[5] in Honduras in which *biweekly* doxycycline was shown to be ineffective in preventing diarrhea. However, many strains of ETEC isolated in this study were resistant to tetracycline, so a definitive conclusion could not be reached about the efficacy of low dose doxycycline.

Despite the evidence that doxycycline, when taken daily, is apparently fairly effective prophylaxis for travelers' diarrhea resulting from sensitive infecting organisms, there are a number of real and potential problems associated with its widespread use:

- Frequent drug use could induce a rising percentage of antibiotic resistant strains of *E. coli* and possibly of *Salmonella* and *Shigella.*
- Since recent studies suggest that both toxigenicity and antibiotic resistance are transferable between organisms, doxycycline use might increase the number of toxin-producing strains as well as the number of multi-resistant strains.
- Several areas of the world already have a high incidence of tetracycline-resistant ETEC. In a recent *Lancet* study,[6] 60 per cent of ETEC isolates from Indonesia, Korea, and the Philippines were tetracy-

cline-resistant. Doxycycline prophylaxis may thus be ineffective in these areas.

- Suppression of normal intestinal flora by antibiotics may result in a proportionally higher incidence of *Salmonella* and *Shigella* enteritis in travelers. Diarrheal illness produced by either of these organisms is frequently more severe, and may be of longer duration than typical ETEC diarrhea.
- Protection lasts only as long as doxycycline therapy is continued.
- Tetracyclines have a number of potentially serious side effects, including nausea, vomiting, and diarrhea. They often are photosensitizers, a particularly troublesome problem in sunny vacation climates.

Given the possible risks and benefits, doxycycline prophylaxis should probably be considered only for selected travelers who plan short-term visits (*i.e.* less than three weeks duration) to high-risk countries without appreciably large tetracycline resistance rates in ETEC isolates. The drug is particularly appropriate for those who require uninterrupted use of their time (*i.e.* for important business transactions) or for those whose general health is fragile. Doxycycline administration must be avoided in patients younger than 8 years and in pregnant women because of the risk of staining teeth.

Pepto-Bismol has been a subject of considerable interest in both prophylaxis and treatment of travelers' diarrhea. The active ingredient — bismuth subsalicylate — has recently been shown to effectively inhibit the activity of cholera and *E. coli* enterotoxins. It is believed that the subsalicylate component exerts an anti-prostaglandin effect, thus "neutralizing" some of the toxin actions. In a double-blind study of 128 American students in Mexico, DuPont[7] showed the efficacy of Pepto-Bismol as a prophylactic agent for travelers' diarrhea. In his trial, diarrhea developed during the period of study in 61 per cent of 66 students taking placebo, compared with 24 per cent on Pepto-Bismol.

One problem with the use of prophylactic Pepto-Bismol in this and other studies is that the doses used were too high for convenient use by most vacationing travelers; *i.e.*, 60 ml four times daily or a bottle a day. A recent in-hospital study,[8] however, demonstrated the effectiveness of nougats (tablets) in preventing diarrhea. Of the 32 volunteers challenged orally with ETEC, only 13 per cent of those taking two tablets of Pepto-Bismol four times daily developed diarrhea, compared with 56 per cent of the placebo group.

Tablet prophylaxis with Pepto-Bismol is easy, convenient, and inexpensive for the traveler. Essentially no adverse effects, including constipation, have been noted to date with this agent, but the vacationer should be warned that the use of oral bismuth will result in black stools and in a black layer of drug coating the mouth and tongue. In addition, the salicylate in Pepto-Bismol could result in toxic levels in those already on salicylates. Serious hemorrhage could occur in patients also taking Coumadin. Finally, a recent study[9] has suggested that concomitant use of Pepto-Bismol may chelate doxycycline, markedly decreasing its bioavailability; hence these two agents should *not* be administered together.

Future steps toward effective prophylaxis against travelers' diarrhea include the development of a polyvalent vaccine directed against the attachment pili of *E. coli*. Still in the early stages of experimental research, this vaccine is designed to induce type-specific antibody which prevents adherence of pili to intestinal mucosa, thus allowing potentially infective organisms to pass harmlessly from the body. A similar experimental vaccine has been effective short-term in preventing diarrheal illness caused by certain strains of ETEC in pigs.

Treatment

The mainstay in treatment of travelers' diarrhea, as with any diarrheal disorder, is rehydration and maintenance of intravascular volume with appropriate electrolyte solutions. In most cases, this can be easily accomplished orally by use of bottled water mixed with foil packets of Gatorade or other glucose/electrolyte preparations especially compounded for athletes. Such ready-mix preparations are convenient and easy to carry in the traveler's luggage. They are tasty, well tolerated under most circumstances, and often in themselves provide substantial symptomatic relief during bouts of diarrhea.

Antimotility agents (Lomotil, Imodium, and paregoric) may relieve cramps and usually reduce the volume and frequency of diarrheal stools. They should be used with caution, however, since the clinical course of some patients may actually be worsened and certain enteroinvasive organisms may become bacteremic with excessive or inappropriate use. Administration of these agents should be limited to three or four total doses during a 24-hour period. They should not be used if stools are bloody or purulent, or if signs of systemic toxicity are present.

Antibiotic therapy is inappropriate for the typical case of travelers' diarrhea, which is usually short-lived and resolves spontaneously. Fresh stool samples for culture and ova/parasite examination should

be obtained in patients with prolonged or severe bouts of diarrhea and in those with bloody stools. Infections due to *Shigella, Campylobacter, Entamoeba* or *Giardia* may require specific therapy if diagnosed by stool examination.

Recent attention has again turned to Pepto-Bismol, this time as a therapeutic rather than a prophylactic agent for travelers' diarrhea. Dupont[10] studied 169 students attending a Mexican university who developed a diarrheal illness, treating them with one of two possible dosage schedules of Pepto-Bismol (either 30 or 60 ml of liquid every 30 minutes for eight total doses) or with placebo. Students in both Pepto-Bismol treatment groups showed a 50 per cent decrease in the number of unformed stools, along with substantial relief of symptoms of abdominal cramping and nausea, when compared with placebo-treated patients. Symptom relief was delayed in onset, beginning after the fourth hour dose. Interestingly, Pepto-Bismol therapy was apparently not effective in relieving symptoms in patients subsequently shown to have shigellosis (primarily an enteroinvasive rather than an enterotoxin-producing organism).

Table I outlines current recommendations for prophylaxis and therapy of travelers'diarrhea.

TABLE I
PROPHYLAXIS AND THERAPY
OF TRAVELERS' DIARRHEA
CURRENT RECOMMENDATIONS

Generally indicated:
(for travel to high risk areas)

- Peptol-Bismol prophylaxis (2 tablets qid)
- Pepto-Bismol therapy for diarrhea (30 cc q ½ hour × 8 doses)
- Glucose-electrolyte ready-mix packets (Gatorade)

Limited indications only:

- Doxycycline prophylaxis (100 mg qd)
- Antimotility agents for diarrhea (< 3-4 doses/24 hours)
- Avoidance of fresh fruits, tap water, restaurants

Not indicated:

- Prophylaxis with Enterovioform, Bactrim, other antibiotics
- Doxycycline prophylaxis for trips > 3 weeks long, non-essential trips, trips in low-risk areas, young children or pregnant women
- Prophylactic antimotility agents (Lomotil, Imodium, paregoric)
- Antibiotic therapy for uncomplicated diarrhea (brief duration, absence of fecal leukocytes, hematochezia, or systemic symptoms)

Summary

In conclusion, the majority of cases of travelers' diarrhea can be prevented by the use of prophylactic Pepto-Bismol. This is most conveniently taken in tablet form in a dose of two tablets 4 times daily for the duration of the trip. Doxycycline is also effective for prophylaxis but should be used only in carefully selected cases.

In travelers who do acquire diarrhea, Pepto-Bismol may attenuate the illness, and therapy with a ready-mix glucose/electrolyte preparation is almost always appropriate. Opiates and anticholinergics should be used cautiously and sparingly. Specific antibiotic therapy is usually reserved for the patient with prolonged, severe or bloody diarrhea in whom a specific infectious agent has been diagnosed by stool examination.

Self-Assessment Questions

Choose the most accurate answer.

1. Of the following, the most important measure in prevention of travelers' diarrhea is:
 a. Drinking only bottled water
 b. Purifying tap water with chlorine tablets
 c. Avoiding restaurant food
 d. Avoiding fresh fruit
2. Clinical features of travelers' diarrhea due to ETEC include:
 a. Presence of fecal leukocytes
 b. Fever and prostration
 c. Secretory diarrhea
 d. Hematochezia
3. Of the following patients, the most reasonable candidate for doxycycline prophylaxis is:
 a. A business traveler on a three day trip to Mexico
 b. A student on a six week trip to Mexico
 c. A business traveler on a three day trip to Norway
 d. A 6-year-old boy on a three day trip to Mexico
4. The most important treatment of travelers' diarrhea is:
 a. Antibiotics capable of eradicating ETEC
 b. Antibiotics capable of eradicating *Salmonella* and *Shigella*
 c. Anti-motility agents, such as Lomotil
 d. Oral fluid and electrolyte replacement

5. Which of the following statements concerning Pepto-Bismol is *false?*
 a. Delayed onset of relief in symptomatic diarrhea (beginning after the four-hour treatment period) is characteristic
 b. Symptoms of salicylate toxicity may occur in patients taking aspirin
 c. Effective prophylaxis is possible if the medication is taken in tablet form
 d. Constipation is a frequent side effect

(Answers on page 464)

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Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

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Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., ***Editor***

Trazodone

Question: Trazodone was recently marketed in the United States. Will you provide general information concerning its indications and use?

Answer: Trazodone HCl (Desyrel) is the first triazolopyridine derivative marketed in the United States for the treatment of depression. Its mechanism is consistent with the biogenic amine theory of depression, which attributes depressive disorders to deficiencies of serotonin or norepinephrine within the central nervous system.[1] Trazodone selectively inhibits the reuptake of serotonin by presynaptic neurons[2] with little effect on norepinephrine.[3, 4] This mechanism differs from prototype antidepressants such as amitriptyline and imipramine. Amitriptyline, more so than imipramine, inhibits the reuptake of serotonin and both agents inhibit the reuptake of norepinephrine.[1] Basic research and clinical studies indicate trazodone has fewer anticholinergic effects than other antidepressants available in the United States.[5-7]

During clinical trials, trazodone was consistently superior to placebo and essentially equal in efficacy to imipramine,[6-9] amitriptyline,[2, 10, 11] and doxepin.[10] Clinically significant improvement was reported in some patients within seven days following treatment with trazodone;[6, 7, 9] however, controlled studies are not available to support this.

Several studies have found trazodone to be better tolerated than other currently available antidepressants.[6, 7, 9, 11] In a review of 58 studies which included 1621 patients, drowsiness (5.6%) was the most frequent adverse effect reported of trazodone.[4] Other adverse effects included tiredness (3.1%), gastrointestinal disorders (2.6%), and dry mouth (2.5%).[4]

Overdosage is a common and dangerous problem with antidepressant drugs. Following a 7.5 gm ingestion of trazodone, one patient experienced only drowsiness and weakness.[4] Thus, this agent may prove less harmful following acute toxicity than tricyclic antidepressants. The median lethal dose in dogs is approximately 500 mg/kg following acute toxicity.[12]

The initial recommended dose of trazodone is 50-150 mg/day.[12, 13] This dosage may be increased by 50 mg/day every three to four days according to patient response.[12] Adult outpatient dosages should not exceed 400 mg/day in divided doses, while inpatient dosages should not exceed 600 mg/day.[12]

Studies indicate trazodone is essentially equal in efficacy, and causes fewer anticholingeric and cardiovascular adverse effects, than previously available antidepressants. Therefore, it may benefit patients unable to tolerate the anticholinergic effects of antidepressants now in use.

Submitted by
Bruce Scott, R.Ph.
Pharmacy Resident

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

The Drug Information Service is maintained by the Department of Pharmacy at the K.U. Medical Center to promote rational drug therapy. The Service is staffed by clinically oriented pharmacists with access to accurate, current, and unbiased information. Information is available free of charge to any health care practitioner involved with patient care. Contact:

Drug Information Service
3930 Cambridge
Kansas City, KS 66103

Telephone (any time): 913-588-2328

The President's Message



Dear Colleague:

The formation of the American Medical Association in 1847 provided the first significant impetus toward improving medical education. The resolution of the New York State Medical Society, which brought together state medical societies and medical colleges to a meeting that initiated development of the AMA, stated:

> WHEREAS, It is believed that a national convention would be conducive to the elevation of the standards of medical education in the United States; and
>
> WHEREAS, There is no code of accomplishing so desirable an object without concert of action on the part of Medical Societies, colleges, and institutions of all states; therefore be it
>
> *Resolved,* That the New York State Medical Society earnestly recommends the National Convention of Delegates for medical societies and colleges in the whole union to convene in the City of New York on the first Monday in May 1846 for the purpose of adopting some concerted action on the subject set forth in the foregoing preamble.

One of the first acts of the organizational meeting of the AMA held in Philadelphia in 1847 was the appointment of the Committee on Medical Education. The first pronouncement of that committee in 1848 reads as follows:

> This association considers defective and erroneous every system of medical instruction which does not rest on the basis of practical demonstration and clinical teaching. It is the duty of medical schools to resort to every honorable means to obtain access for their students to the wards of a well-regulated hospital.

From this background, the AMA has continued to be involved in medical education at all levels. The committee struggled with the elimination of "diploma mills" and providing sound education for future physicians during the next 50 years. In 1904, the name of the Committee on Education was changed to the Council on Medical Education, and that Council still exists to this date.

Soon after its establishment, the Council set up standards for pre-medical education, undergraduate medical education, establishment of the internship and, later, for residency training. These ideals advocated adequate training in the basic sciences followed by clinical experience that included direct patient contact. The standards are responsible for the excellent system of medical education that we enjoy in the United States today.

As Kansans, we are proud of the election of William A. Reals, M.D., Dean, The University of Kansas School of Medicine-Wichita, to the prestigious Council on Medical Education. Our initial success came in filling an unexpired term at Las Vegas in December 1981. We were again successful when he was reelected to a full three-year term on the Council in June 1982. Since that time, the Council has completed and published *Recommendations for Future Directions for Medical Education.* This report is worth reading because it outlines the direction in which education should go and emphasizes its position, *i.e.,* to train physicians first, and then specialists.

Education is the backbone of any profession; it provides physicians with the training to serve patients to the best of their ability, based on scientific fact and clinical experience. Therefore, it is imperative that all physicians support the organization that deals with such a critical part of their own being — past, present, and future.

For those who are AMA members, we thank you for your support. For those who are not members, we urge your reconsideration, for the education mission itself is worth the price of membership.

President



The Battered Student Syndrome

Generations do not exist in explicit terms. They are the product of daily and consequently subtle progression, but we are disposed to speak of them as finite entities as though the sun were some massive strobe light which holds them in static image to serve the convenience of communication. Thus, with more temporal purpose than accuracy, we speak of the older generation, the younger, the lost, the wild, and so on. And it is the first two we speak of now.

Contrary to Thomas Wolfe's pessimistic admonition, you can go home again but the neighborhood has changed. The known faces are no longer there — at least as you remembered them — and distressingly few remember you. This somber conclusion is the result of pondering the medical education experience of a good many years ago as it has come to mind with the increasing publicity being given to the complaints of the younger (see above) generation that they are being mistreated.

It is, of course, impossible to interpret the earlier experience in the exact terms of the current one. Whatever the superficial similarities and intrinsic identities, the extrinsic factors are too different. Medical education has changed through the more than 40 years we look back upon — all education for that matter — despite the persistence of certain basic features. (Incidentally we note that they are still advising premedical students to take more diversified courses rather than concentrating so heavily on science in the hope it will expedite admission to medical school, so that's *one* thing that hasn't changed.) But education is a constantly changing process — not that the young create the changes — they are created by mankind's insatiable curiosity and the young become the agents of effect on their way to meeting the changes in *their* time.

Perhaps ours were simpler days — we didn't think so at the time but the young of today undoubtedly do. In approaching a medical career, we (both personal and collective) assumed there would be more severe demands in medical education to prepare for the comparable demands of professional life. It was a deeply traditional and authoritative world, the patterns of academic behavior were consistent with what we had heard and read about the life and if we felt misused, it could hardly be called an unexpected experience, saying as much about us as the system. Gaining entrance to medical school was a minor miracle, not without some lingering fear that the Admissions Committee might follow up their first telegram with another saying sorry, it was all a mistake. Once we were on the premises, this gave way to a dedication to remaining, a condition that subsided only gradually as the passage of time and testing landmarks led to the cautious hope that we would really make it all the way. In the process, students complained frequently and fervently about the methods — but to each other. Perhaps we could have bearded the academic lions in their dens and survived but we didn't know we could and rarely had the temerity to try. Demonstrations — strikes — unions — totally foreign thoughts applying to other worlds.

So we acknowledge the persistence of this generational exercise and add to it our certainty that the medical profession is one of the more dependable participants in this game of comparing the component age groups, a condition in exacerbation at the moment. Specifically, the treatment of embryonic and fetal physicians during the prenatal years is getting poor marks from some of the subjects, having been seriously compared in form and effect with child abuse.

Not unpredictably, this has stimulated varied reactions. Personally, we find it repugnant to put the distresses of medical students in the same category as the tragic and destructive effects of child abuse despite semantically contrived similarities. Even if the psychopathology and manifestations of child abuse had been as well recognized in those days as now, we doubt that it would have occurred to us to see our experiences in such a light. This is not to

claim a greater virtue than now exists but the differences in the two conditions are far more numerous and significant than any similarities. If nothing else, we had the prospect of a day of release coming up, a situation not, it would seem, apparent to the abused child. Meantime, there was work to be done and we made full use of that extracurricular but essential course of student life, the principles and practice of griping, without which no one could graduate and which proved invaluable for that activity most of us would embark upon after graduation — military service. Despite the many changes in these 40 years to prepare today's students for a very different professional world, we hope that hasn't changed (and in fact, judge that this whole thing is an exercise of that fundamental activity).

This is certainly not to imply that our days were unalloyed pleasure — even including the pleasure of shared misery. We encountered what we felt to be our fair share of annoying individuals in the academic gowns. In retrospect, we still agree with our assessment of the moment — that some were certified, dyed-in-the-wool stinkers. Some proved later to be friendly and enjoyable people whose academic personalities seemed somewhat like costumes they put on for the pedagogic occasion. Some — particularly in the preclinical area — were so engrossed in their work that the students were not depersonalized so much as impersonalized — a necessary nuisance but ranking somewhere between the laboratory animals and other apparatus necessary to accomplish the full purpose. And some of our assessments were, we see now, simply our misinterpretations of the circumstances born of our own anxieties. The system itself did provide the atmosphere since there was no question but that it was designed to keep the victims impressed with the serious nature of the whole thing.

But abuse in the sense of degrading or lasting damage? Maybe we were too obtuse to recognize it as such. It seemed that it was simply the realization of the expectation — the encountering of the anticipation — and if we had trouble enjoying parts of it, we presumed that was the nature of the thing and it was up to us to accommodate. Maybe we were shortsighted — so preoccupied with the process we failed to appreciate that we should have been assaulting the system on behalf of generations to come if not ourselves. If it seems we were unhealthily docile or passive, it reflects our lack of realization that we were prisoners of an abusive system although by definition we must have been. This brings up the acknowledged etiologic element in child abuse — the fact that the offender was himself abused in childhood. If the premise is valid, then, that students of today are abused, it follows that those of us from that period — at least the academic colleagues from our ranks — gained qualification from our own mistreatment as our abusers did from theirs and so ad retroinfinitum (a word that could be coined only by an abused mind).

One aspect of the situation does seem to have been neglected, however — the impact of such attitudes and methods in enhancing the various expressions of discrimination, mainly the racial and sexual. It can be said — in explanation, not expiation — that the academic discriminatory practices of that day simply reflected the general patterns of society. From the racial standpoint, it can also be said — and it may raise a few hackles — that it fostered a ghetto philosophy since the restrictions of the predominantly white and gentile schools tended to divert these ethnic groups to institutions catering to their own. There were no blacks in our class (and we wouldn't have called them blacks since that would have been disrespectful in those days) so it was easy to ignore that concern. The Jewish group was close-bound inside the classroom and out. The relationship was friendly but again the segregation was not so much physical as social and reflected the public attitude.

Much the same can be said for women. They were accepted on personal terms of friendship but their cause was by no means championed — or even seen as a cause. The roles of women as students and practitioners was determined by personal responses on both sides, we assumed — not an established policy of prejudice. If they elected to pursue medical careers, their specific problems were part of it. Objections to women in medicine were of stock variety — they wasted the academic effort since they wouldn't remain in practice, they weren't physically capable of meeting the demands, they weren't emotionally equipped to handle the pressures, male patients would never accept them, and so on. The obvious errors in such thinking have been demonstrated repeatedly and are on their way to elimination as the increased enrollment of women testifies. Still, such were the general thoughts of the day — we didn't create them but neither did we see it as our purpose to make drastic changes. (And incidentally, in our class, the general success level of that group has well exceeded that of the male segment.)

But this interjection is not to turn to the problem of discrimination but to note that if the medical education process is demeaning, how much more so to these for whom it is an added indignity? One cannot ponder the problem at any length without coming to

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AMA House of Delegates

(Continued from page 393)

Future Directions for Medical Education

This comprehensive report contained 36 recommendations culminating a two-year effort involving six task forces. One major proposal approved by the House called for a revival of the general internship as the transitional year between medical school and residency.

Other topics included generalism and specialism; preparation for and admission to medical school; continuing medical education; and licensure for the practice of medicine.

Federal Financial Aid Programs for Medical Students

In a two-part substitute resolution the House voted: That the AMA continue to urge the President and the Congress to maintain the eligibility of medical students to participate in the Guaranteed Student Loan Program; and that the AMA continue to urge the President and the Congress to fund direct student loan programs available to medical students in amounts equal to or greater than those of fiscal year 1982 for all such programs.

Corporation or Employer-Sponsored Examinations

The House approved a policy stating that the AMA encourages employers who provide or arrange for special or comprehensive medical examinations of employees to be responsible for assuring that these examinations are done by physicians competent to perform the type of examination required. Whenever practical, the employee should be referred to his/her personal physician for such professional services. In many instances in which an employee has no personal physician, efforts should be made to assist him/her in obtaining one, with emphasis on continuity of care. This effort should be aided by the local medical society whenever possible.

Pension Reforms

The House voted that the AMA continue to support equity in pension plans, including appropriate increases in the maximum contribution to self-employed retirement plans (Keogh), and actively oppose enactment of H.R. 6410, the Pension Equity Tax Act of 1982, as introduced because of its major discriminatory provisions.

Civilian-Military Contingency Hospital System

The House supported the System, which will enable the United States to prepare for the treatment of casualties from any future conventional military conflict.

Medical Consequences of Nuclear War

The House approved a report detailing several AMA activities pertaining to the medical consequences of nuclear war. In addition, the House called for a study of the medical effectiveness of relocation and shelter plans proposed by the Federal Emergency Management Agency.

Designation of Areas of Medical Need

The House passed a resolution stating that the AMA should ask that the federal government consolidate the designation process for identifying areas of medical need; coordinate designation processes with state agencies to obviate duplicative activities; ask for state and local medical society approval of designated underserved areas; and continue the rules requiring automatic cessation of federal subsidies when manpower guidelines are met.

Insurance Assignments

The House authorized the AMA to investigate the frequency of and seek a solution to the problem of erroneous payments to insurance beneficiaries instead of physicians to whom they have assigned such payments.

State Regulation of Professional Liability Insurance

The House supported the current system of state regulation of professional liability insurance and opposed any effort by the federal government to pre-empt this right of the states.

Tobacco and Health

The House reaffirmed its support for the concept of rotational warnings on cigarette packages; it also supported the concept of a federal Office on Smoking and Health.

Smoking Related Fires

The House voted to support the concept of self-extinguishing cigarettes; to continue support for "The Cigarette Safety Act" calling for a study to determine the feasibility and practicability of establishing a standard for self-extinguishing cigarettes and requiring cigarette manufacturers to meet that

standard; to inform major cigarette manufacturers of its support of the concept of self-extinguishing cigarettes for the purpose of reducing fire related deaths, injuries, and loss of property; and to reiterate opposition to all smoking.

Third Party Coverage for Psychiatric Services

Upon being informed of the continuing disparity of third party coverage for psychiatric services, the House reaffirmed AMA support for equivalent benefits.

Automobile Deaths and Injuries

The House supported state legislation mandating the use of seatbelts and requiring restraint of infants and children in motor vehicles.

Scientific Reports

The House considered a number of reports from the AMA Council on Scientific Affairs. Topics included medical evaluations of healthy persons; sodium in processed foods; continuous ambulatory peritoneal dialysis; infant formula marketing; fetal effects of maternal alcohol use; and brain injury in boxing.

We, your Delegates, urge you to let us know your opinions so that we may represent you better. Even if you cannot attend the meeting personally, you can prepare a resolution and request that it be submitted to the House by one of your official Delegates. The next AMA House of Delegates meeting will be the 1982 interim meeting in Miami, December 5-8, 1982.

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Kenneth E. Hedrick, MD, R
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Charles Stephens, MD, S
Thornton L. Waylan, MD
Chang S. Yoon, MD, R

Vox Dox

(Continued from page 396)

tion recall at a point in time is measured. Again other parameters of knowledge and skill need consideration.

It is time to assess what we support with big dollars and requirements for licensure. Let us have the insight to know what is helping us accomplish the goal of better patient care, the strength to reject what is not, and the wisdom to know the difference.

Arthur C. Cherry, Jr., M.D.
918 West Tenth
Topeka KS 66604

Vox Dox Editor:

The St. Paul-Ramsey Medical Education and Research Foundation has been actively involved in compiling a resource directory for physicians with physical disability. The purpose of the project is to form a voluntary group of physicians to provide information and referral services as well as support and advocacy to physicians who incur the same disability and need specific information. I became deaf seven years ago just as I was completing medical school and can readily attest to the paucity of information available to this unique population. Existing rehabilitation programs are simply not equipped to deal with the situation.

The biggest problem we are encountering is that of identification. It is currently estimated that 4 per cent of all physicians are not in active practice because of a physically disabling condition, and that 25 per cent of these physicians have the potential to be rehabilitated into the active practice of medicine. In real numbers, this constitutes 1 per cent of the licensed physicians in this country, or 4,500 physicians. Our goal is to identify these physicians and to encourage their participation. To date we have placed advertisements in more than 100 major medical journals; we have received responses from fewer than 200 physicians.

In retrospect, it appears this was due to use of inappropriate terminology in the ads. Physical disability does not imply inability. My use of the term "handicapped physician" was a misnomer since the majority of physically disabled physicians are not handicapped in their practice of medicine. I apologize for the inappropriate terminology and again ask that all physicians, active or inactive, with any type of physical disability contact me at the address below. We anticipate that the directory will be completed in six to eight months, and at that time it will be sent only to those physicians who are listed therein. Upon receipt of your initial response, information forms will be mailed.

All physicians with physical disability, no matter how small, are encouraged to respond. Information from a physician with even a minor disability may be of value to another with multiple disabilities. The cornerstone of this project is your participation.

Frank C. Zondlo, M.D.
St. Paul-Ramsey Medical & Educational Research Foundation
640 Jackson Street
St. Paul MN 55101

The Battered Student Syndrome

(Continued from page 454)

the conclusion that it is a deeply personal matter — not so much the system or any organized effort — age, academics, sex, race, or whatever — in which the contentions between the individual actors is basic. If there is a totality of effect which can be attributed to the system, it is only because the individuals — faculty and students — pursue primordial drives which condition the effort and response. If it was abusive, so has the world been.

Although the methodology is somewhat different, we are inclined to view the whole matter as a contemporary rendering of the age-old (and thus, like generations, continuing) ritual: the assault on the tribal chiefs by the young bucks (and some does) who seek to displace them. The old ones, longing for immortality, have begotten the young only to find to their chagrin that the relentless process presses them on to their termination. They confront the threat by attempting on the one hand to control and restrict and on the other to inspire and develop. If the system works out to seem demeaning to the young, it may indicate a willful ineptitude on the part of the seniors — even lack of memory or imagination — but we recommend to the students a policy of patience and forbearance. They will shortly move into the positions of authority and will find that with startling suddenness, time will pass and they will be the seniors with all the rights and privileges — and complaints of the new young — thereof. — *D.E.G.*

dis·ease

/diz-ez/ 1. Chronic dependency upon alcohol; impairing the performance of vital functions. 2. Arrested through effective alcoholism treatment program.

Chances are, during a patient's preliminary examination, alcoholism is not one of your immediate suspicions. But with 10 million Americans suffering from this disease, and over 100,000 in the Kansas City area alone, perhaps it should be. Or perhaps the drinking problem is someone within your patient's family, causing them mental and even physical abuse.

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If you have a patient with a drinking problem, send for our detailed informational brochure, or personally contact one of our professional staff. Outpatient: Paul Anderson, Ph.D., Family Institute of Addiction Treatment, 7840 Washington Ave. Suite 300, K.C., KS. 66112. (913) 334-6300. Inpatient information: Sue Little, Director, Bethany Medical Center, 51 North 12th Street, K.C., KS. 66102. (913) 281-8951.

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KaMPAC
Kansas Medical Political Action Committee

KaMPAC is a voluntary, non-profit, unincorporated committee of individual physicians and others, and is not affiliated with any political party.

The purposes of the Committee are:

1. To promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective role in governmental affairs;
2. To encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of office holders, and the pertinent qualifications of candidates for elective office; and
3. To assist physicians and others to organize for effective political action to promote civic responsibility and integrity in government.

KaMPAC operates independently of the Kansas Medical Society. KMS contributes to KaMPAC's educational programs, but cannot by law contribute to its candidate support activities.

You can help organized medicine have an impact on the legislative process by joining KaMPAC today.

WHATEVER YOUR POLITICS . . . VOTE KaMPAC!

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Mail to KaMPAC, 1300 Topeka Avenue, Topeka, KS 66612.

* May designate KaMPAC only

Smallpox Vaccinations

The worldwide eradication of smallpox in 1977 eliminated the last remaining need for smallpox vaccinations for the general public, the AMA reports. Yet, according to the Center for Disease Control (CDC), more than 2.8 million doses of smallpox vaccine were distributed in 1980 in the United States alone, some of which are being used inappropriately. One such case, reported by Lawrence Mintz, M.D., in *JAMA* involves use of the vaccine to treat a patient with recurrent genital herpes simplex viral infection. The vaccine not only failed to cure the original infection, it produced a new area of herpes lesions at the inoculation site on the upper arm. Smallpox vaccination per se carries a risk of complications which occur at a rate of about 135/million vaccinations. J. Michael Lane, M.D., M.P.H., director of the CDC's Center for Prevention Services, believes that smallpox vaccination is worthless and dangerous as a treatment for herpes simplex virus infection. According to Lane, the only reason to use smallpox vaccine today is to protect laboratory workers exposed to smallpox and related vaccines. He concludes, "I have one word of advice for those who continue to use Jenner's wonderful vaccine. *Stop*."

Physician Services

The AMA's Socio-economic Monitoring System has to date conducted two quarterly surveys, the final quarter of 1981 and the first quarter of 1982. Results of the survey indicate: (1) A general decline in the average number of services performed per week. In the "All Physicians" category, three out of four service classifications declined in number of weekly services. (2) Decline in surgical visits. (3) Prices for medical and diagnostic procedures have increased appreciably while surgical fees have increased moderately. (4) In the Medical and Diagnostic category, total expenditures fell for newborn care (−31.9%) and childhood annual exams (−9.4%). Largest increase was for new patient office visits (18.2%). In the Surgical category, expenditures rose for five of the seven procedures reported. However, there was a sharp decline in expenditures for total hysterectomies (−19.6%).

SCIENTIFIC/SKI MEETING: The Northwestern Medical Association convenes for its 36th Annual Meeting at Sun Valley, ID, from February 7 to 11, 1983. Transplants-implants, general medical subjects, ski-injury prevention, high-altitude physiology and financial planning will be discussed by experts. Approved for 10 CME Category I credits. Registration 3 to 5 p.m. on February 7, Challenger Inn, Sun Valley. Non-members registration $100. For information, write to Norman Christensen, M.D., 2456 Buhne Street, Eureka, CA 95501 or phone (707) 443-2248.

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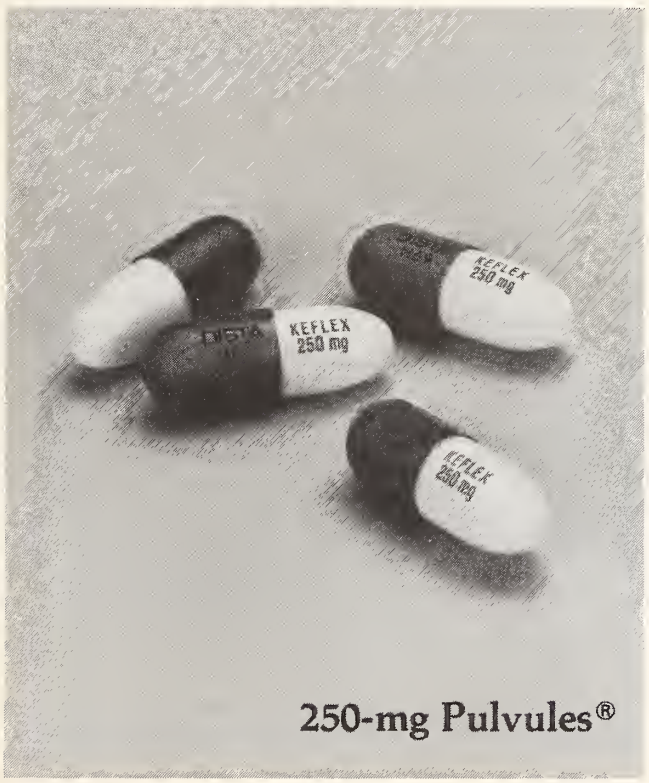
The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

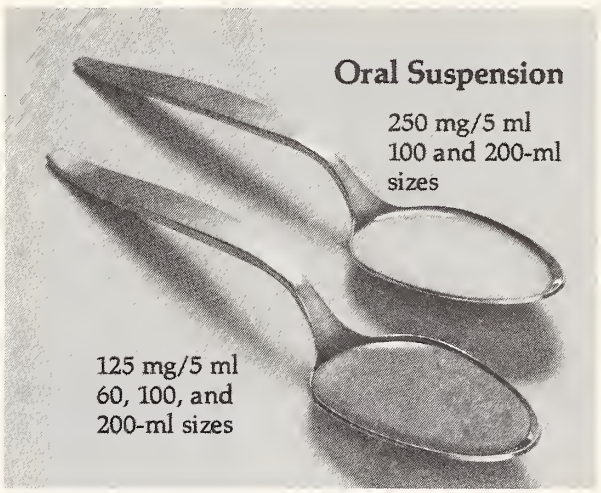
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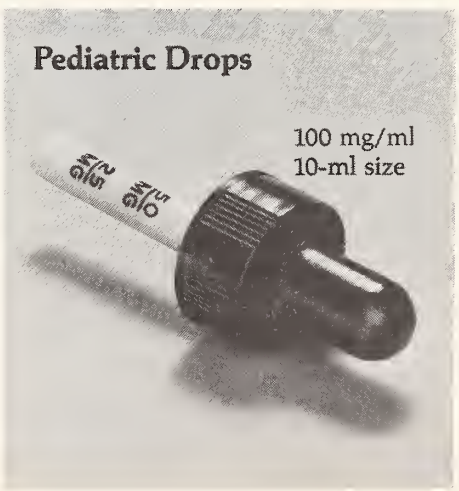
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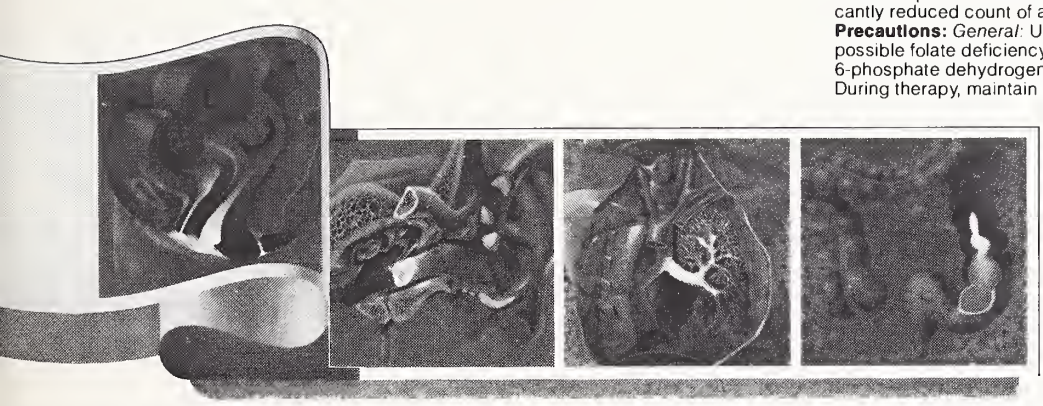
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For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.
For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.
For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.
Also for the treatment of documented *Pneumocystis carinii* pneumonitis.
Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.
Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.
Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.
Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.
Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.
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Dosage: Not recommended for infants less than two months of age.
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Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.
Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.
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1. Rubin RH, Swartz MN: *N Engl J Med* 303:426-432, Aug 21, 1980. 2. Data on file, Medical Department, Hoffmann-La Roche Inc.

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OCTOBER
1982

(ISSN 0022-8699)

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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KMS President Visits Council Districts

Each year it is customary for the new KMS President to visit the Council Districts in order to review the upcoming year's work and to answer questions concerning KMS activities.

In addition to Kermit G. Wedel, M.D., President, those attending the meetings this year include Steve Carter, Executive Director; Roger D. Warren, M.D., KaMPAC Chairman; and a representative from the University of Kansas School of Medicine.

Each local member physician is encouraged to attend his/her Council District meeting.

COUNCIL DISTRICT MEETING SCHEDULE

District	*Date*	*Location*
1	October 20, 1982	Atchison
2	October 19, 1982	Kansas City
3	October 26, 1982	Shawnee Mission
4	January 25, 1983	Pittsburg
5	September 15, 1982	Manhattan
6	June 7, 1982	Topeka
7	November 2, 1982	Emporia
8	October 21, 1982	Winfield
9	November 16, 1982	Salina
10	November 23, 1982	Halstead
11	January 4, 1983	Wichita
12	October 27, 1982	Medicine Lodge
13	November 18, 1982	Hays
14	October 13, 1982	Great Bend
15	October 28, 1982	Liberal
16	October 6, 1982	Colby
17	November 9, 1982	Garden City
18	January 18, 1983	Lawrence
19	November 17, 1982	Independence

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WARNINGS

CARDIAC FAILURE: In congestive heart failure, inhibition with beta-blockade carries the potential hazard of further depressing myocardial contractility and precipitating cardiac failure. In patients already receiving digitalis, propranolol may reduce the positive inotropic action of digitalis and may have an additive depressant effect on AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, in rare instances, cardiac failure has developed during propranolol therapy. At the first sign of impending cardiac failure, patients should be fully digitalized and/or given a diuretic, and observed closely: a) if cardiac failure continues, despite adequate digitalization and diuretic therapy, propranolol should be immediately withdrawn; b) if tachyarrhythmia is being controlled, patients should be maintained on combined therapy and closely followed until threat of cardiac failure is over.

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuation of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when INDERAL is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease, who are given propranolol for other indications.

IN PATIENTS WITH THYROTOXICOSIS, possible deleterious effects from long term use have not been adequately appraised. Give special consideration to propranolol's potential for aggravating congestive heart failure. Propranolol may mask the clinical signs of developing or continuing hyperthyroidism or complications and give a false impression of improvement. Propranolol should be withdrawn slowly, since abrupt withdrawal may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

IN PATIENTS UNDERGOING MAJOR SURGERY, beta-blockade impairs the ability of the heart to respond to reflex stimuli. Except in pheochromocytoma, propranolol should be withdrawn 48 hours prior to surgery. In case of emergency surgery, the effects of propranolol can be reversed by administration of beta-receptor agonists such as isoproterenol or levarterenol, but such patients may be subject to protracted severe hypotension. Difficulty in restarting and maintaining the heart beat has been reported.

IN PATIENTS PRONE TO NONALLERGIC BRONCHOSPASM (e.g., CHRONIC BRONCHITIS, EMPHYSEMA), administer with caution, since propranolol may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta-receptors.

DIABETICS AND PATIENTS SUBJECT TO HYPOGLYCEMIA. Propranolol may prevent the appearance of premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia, especially in patients with labile diabetes. A precipitous elevation of blood pressure may accompany hypoglycemic attacks.

USE IN PREGNANCY: Safe use in human pregnancy not established. Embryotoxic effects have been seen in animals at doses about 10 times the maximum recommended human dose.

PRECAUTIONS

Patients receiving catecholamine depleting drugs such as reserpine should be closely observed if propranolol is administered, since it may occasionally produce hypotension and/or marked bradycardia resulting in vertigo, syncopal attacks, or orthostatic hypotension.

Observe laboratory parameters at regular intervals. Use with caution in patients with impaired renal or hepatic function.

ADVERSE REACTIONS

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; arterial insufficiency, usually of the Raynaud type; thrombocytopenic purpura. *Central Nervous System:* lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. *Gastrointestinal:* nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis. *Allergic:* pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress. *Respiratory:* bronchospasm. *Hematologic:* agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura. *Miscellaneous:* reversible alopecia. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta-blocker (practolol) have not been conclusively associated with propranolol. *Clinical Laboratory Test Findings.* Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

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—Each hexagonal-shaped, yellow, scored tablet is embossed with an "I" and imprinted with "INDERAL 80," contains 80 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0428-81) and 1,000 (NDC 0046-0428-91). Also in unit dose package of 100 (NDC 0046-0428-99).

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Store at room temperature (approximately 25° C).

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—Each ml contains 1 mg of propranolol hydrochloride in Water for Injection. The pH is adjusted with citric acid. Supplied as 1 ml ampuls in boxes of 10 (NDC 0046-3265-10).

Store at room temperature (approximately 25° C).

7997/882

Reference: 1. Freis, E.D.: Hypertension (Suppl. II) 3:230 (Nov.-Dec.) 1981

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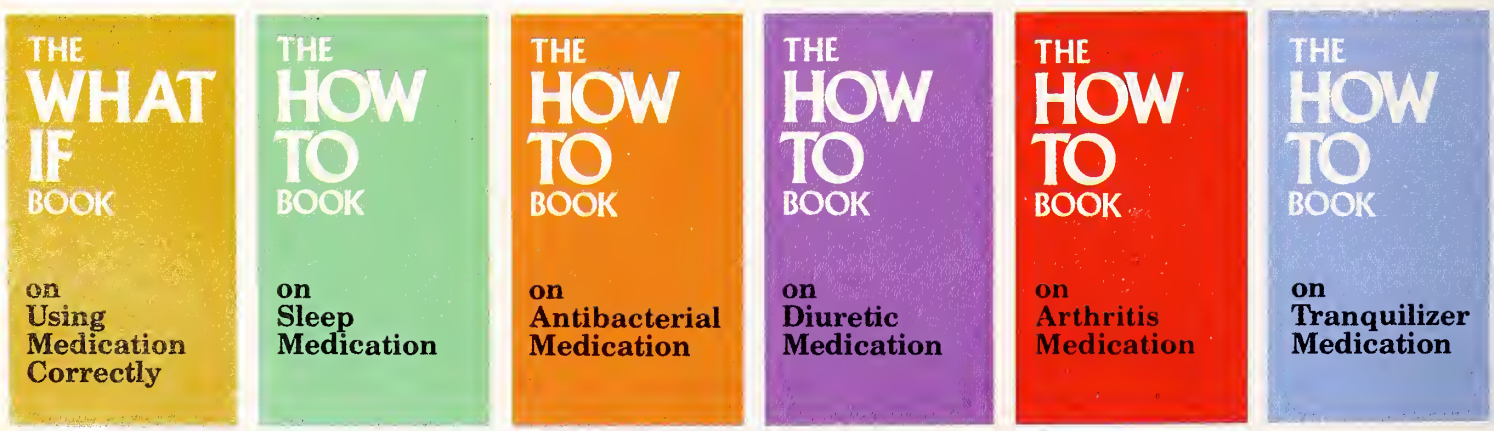
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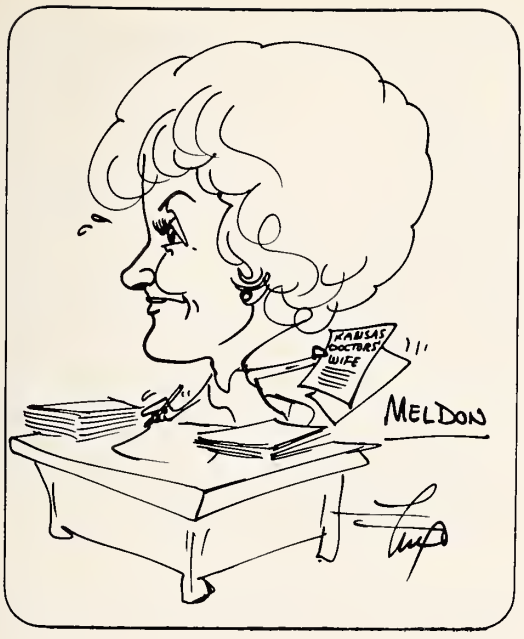
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

In addition to the ongoing projects to which we are giving our time and energy (AMAERF, legislation, international health, membership, etc.), this year we are focusing on two areas: drug abuse and public communications.

Our Auxiliary has had a great deal of help as well as moral support from the Texas Medical Auxiliary in organizing our campaign against drug abuse. We in Kansas do not have the financial resources that were available to the Texas Auxiliary, but we do have STOP POT bumper stickers, which we are selling at cost — four for $1.00 (mail orders three for $1.00 from KMS headquarters). We hope every physician's family in Kansas will use STOP POT bumper stickers on their cars and give or sell others. We will also be assisting the Kansas Medical Society with your Drug Forum scheduled for April 8 and 9 in Salina. Nancy Roderick (James) is our Drug Abuse chairman and is assisting Dr. Wedel on that project.

In spring 1981, KMS asked the help of our Auxiliary in initiating a "Professional Communications Program." The program was originally conceived by the Burroughs-Wellcome Company, and has been adopted by a number of state medical societies with good results. Staff from the Burroughs-Wellcome Company held a training session for 24 of our members and provided an exceptionally good kit of questions to ask each physician on the subject of his/her choice. The program has been well received by both the public and the physicians participating. This year we hope to have more implementation of this program, and ask your cooperation in participating when one is organized in your county. There are 84 radio stations in Kansas. In most counties, an Auxiliary member sets up the five-minute taped interview, and also is the interviewer. In one county, the radio station provides a broadcaster as interviewer and the program is done live. We believe these programs provide a good public relations opportunity for the medical profession, as well as public education. Lela May Young (Chester) of Kansas City is chairman of Radio Communications.

Meldon Laury
President
Kansas Medical Society Auxiliary

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

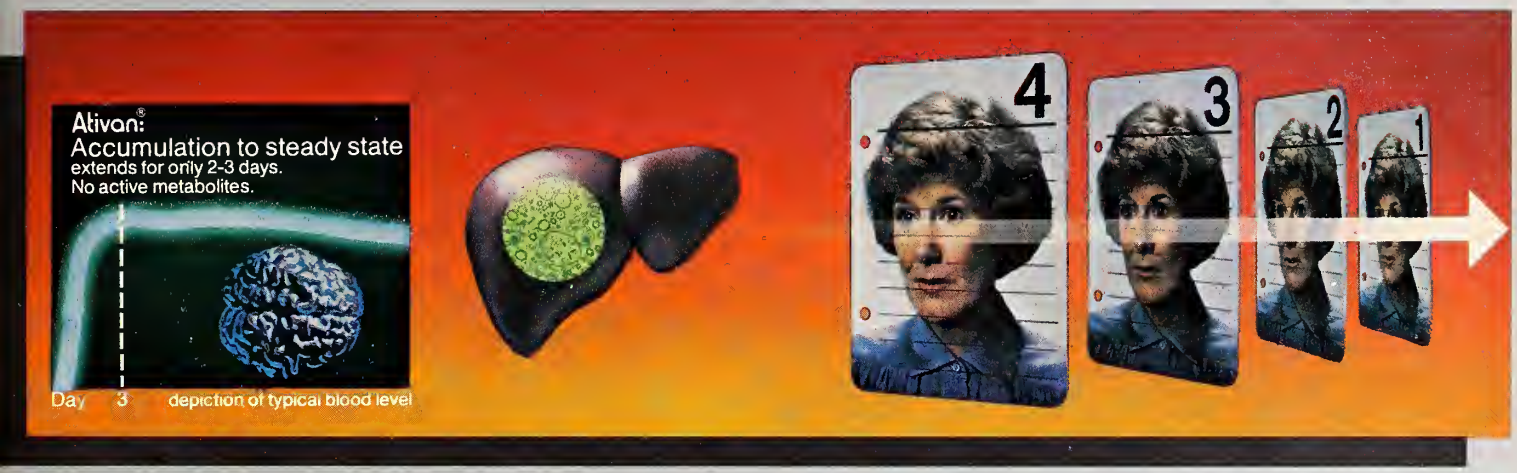
Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

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Unlike most benzodiazepines, Ativan has a short half-life, no active metabolites, and accumulation to steady state extends for only 2-3 days. Ativan is therefore less likely to cause excessive sedation.*

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The short half-life of Ativan facilitates more rapid response to dosage adjustments, aiding you in titrating therapy to the patient's changing needs. Once you decide to discontinue Ativan, it will be out of your patient's system 4 days after the final dose—unlike the long-acting, multi-metabolite benzodiazepines which take as long as 2 weeks to be totally eliminated.

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References:
1. Klotz U, Reimann I: N Engl J Med *302*:1012-1014, 1980.
2. Desmond PV, Patwardhan RV, Schenker S, et al: Ann Intern Med *93*:266-268, 1980.
3. Patwardhan RV, Yarborough GW, Desmond PV, et al: Gastroenterology *79*:912-916, 1980.
4. Sellers EM, Naranjo CA, Peachey JE: N Engl J Med *305*:1255-1262, 1981.
5. Ruffalo RL, Thompson JF, Segal JL: South Med J *74*:1075-1078, 1981.

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*Pharmacokinetics cannot as yet be directly related to efficacy.
†All benzodiazepines produce additive effects when given with CNS depressants such as barbiturates or alcohol.

See important information on following page.

Ativan® for (lorazepam) IV Anxiety

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan®
for (lorazepam) IV
Anxiety

Dosage: **Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.**

How Supplied: **0.5, 1.0 and 2.0mg tablets.**

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Vox Dox

Vox Dox Editor:

I would like to express my deep gratitude for your "Consultation Piece" in the Editorial section of *The Journal of the Kansas Medical Society* (June 1982).

Speaking for the pathologists in the state, I am in complete agreement with your editorial comment. The article demonstrated great insight into the problem of insufficient clinical information (a problem we all face on numerous occasions daily), and your touch of humor was very effective in getting the point across.

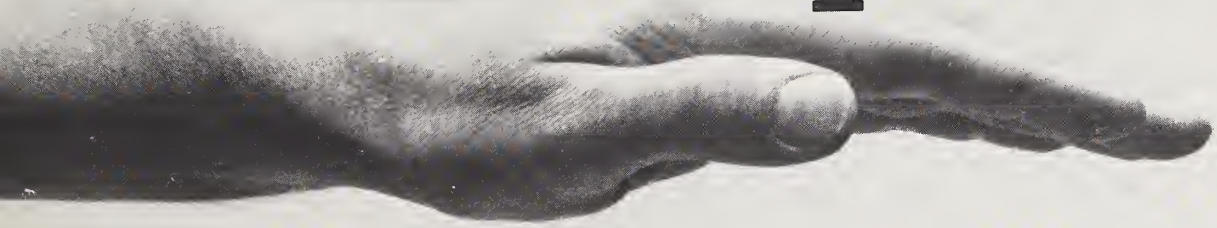
I only hope that a sufficient number of clinicians read the comment and take it to heart. Let's face it — many pathologists have a bit of the clinician in them. We would be delighted at the opportunity to be consulted in a variety of clinical situations, where the full impact of the advances in laboratory medicine would be brought to bear for the benefit of the patient. What a great stride forward it would be in the delivery of health care! How tremendously cost-effective it would be! How much more effectively we could help the clinician resolve the myriad of "rule-out" admitting diagnoses, if we could only be provided with the appropriate clinical information.

Roman Hiszczynskyj, M.D.
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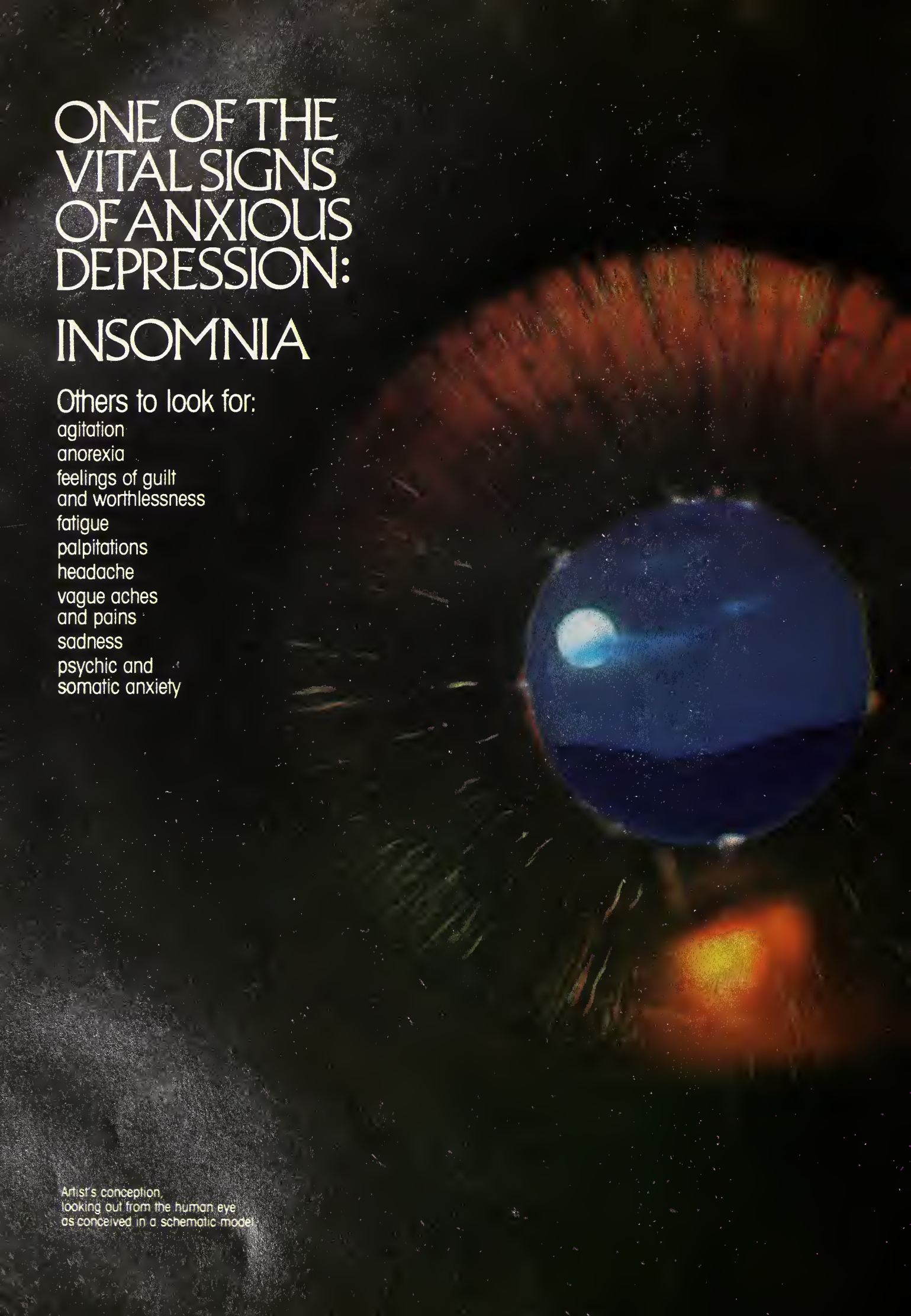
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INSOMNIA

Others to look for:

agitation
anorexia
feelings of guilt and worthlessness
fatigue
palpitations
headache
vague aches and pains
sadness
psychic and somatic anxiety



Artist's conception, looking out from the human eye as conceived in a schematic model.

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in moderate depression and anxiety

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Please see summary of complete product information on following page.

LIMBITROL® TABLETS Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose* packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50.

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KaMPAC MEMBERS 1982

The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

Membership Categories

Certificate	$500—C
Sustaining	100—S
Regular	60—R

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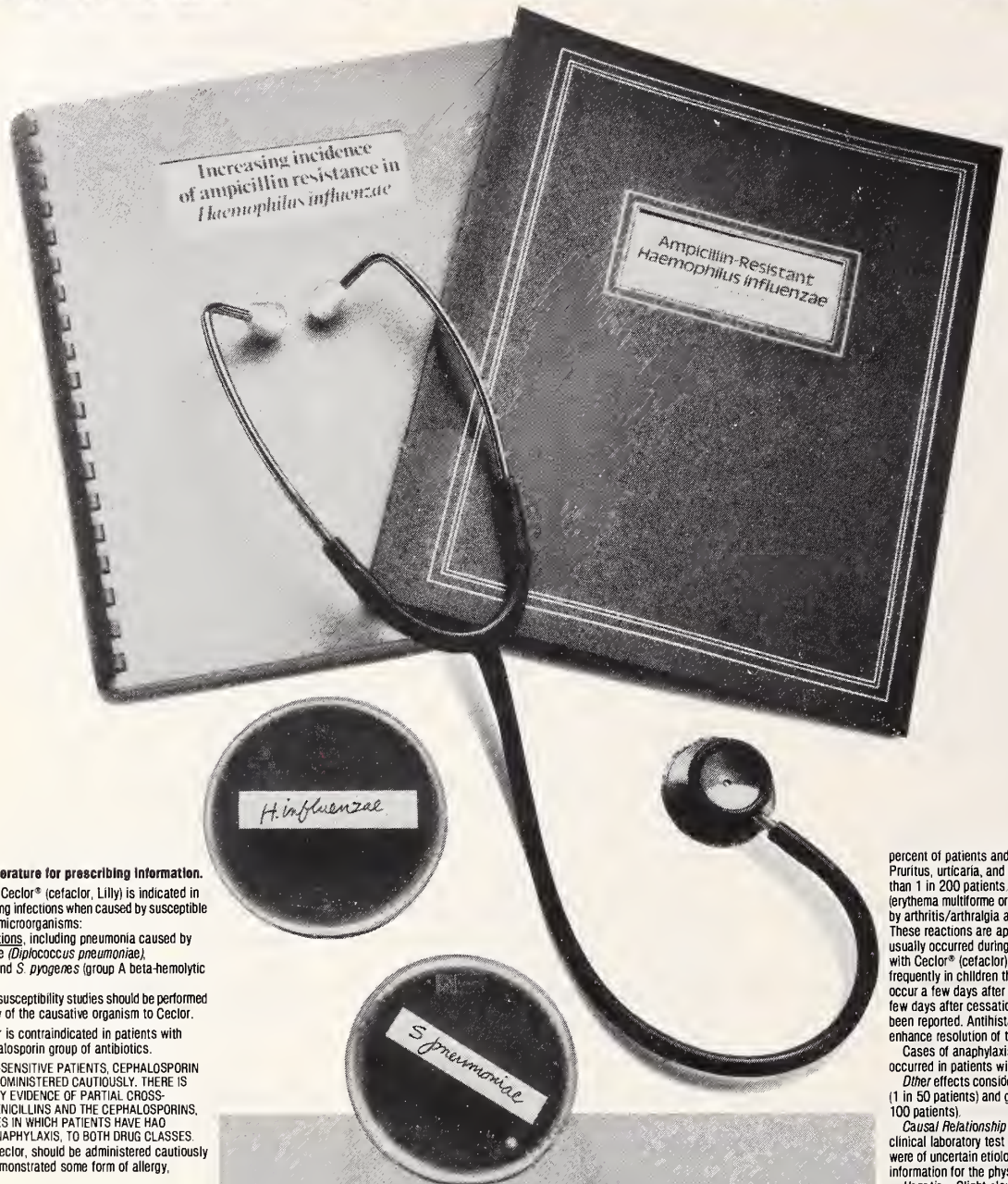
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(Continued on page 516)

An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

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Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coomb testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: Adverse effects considered related to cefaclor therapy are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70) and nausea and vomiting (1 in 90).

As with other broad-spectrum antibiotics, colitis, including rare instances of pseudomembranous colitis, has been reported in conjunction with therapy with Ceclor.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor® (cefaclor). Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [100281R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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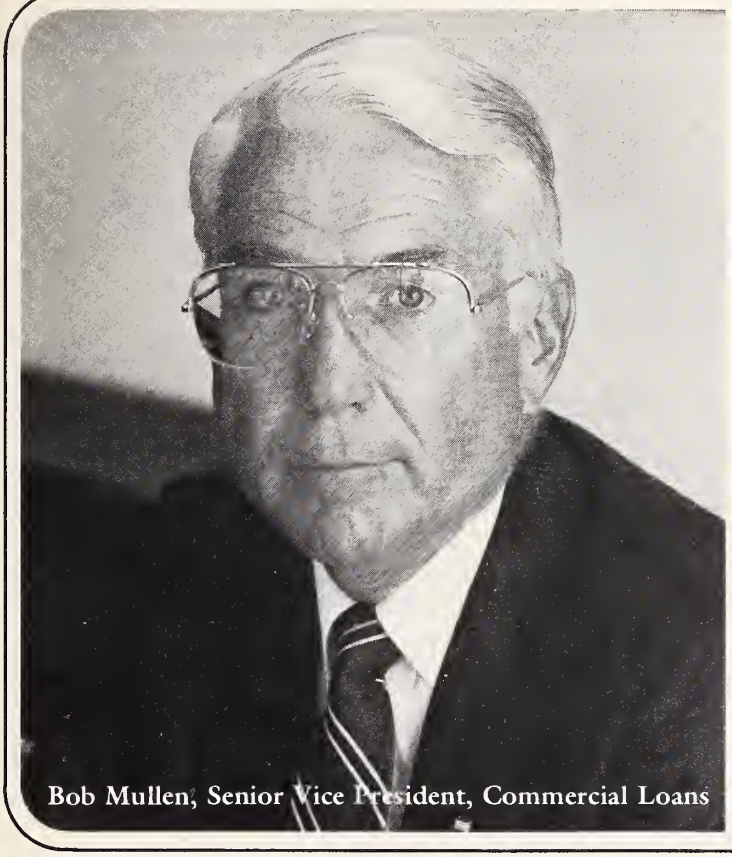
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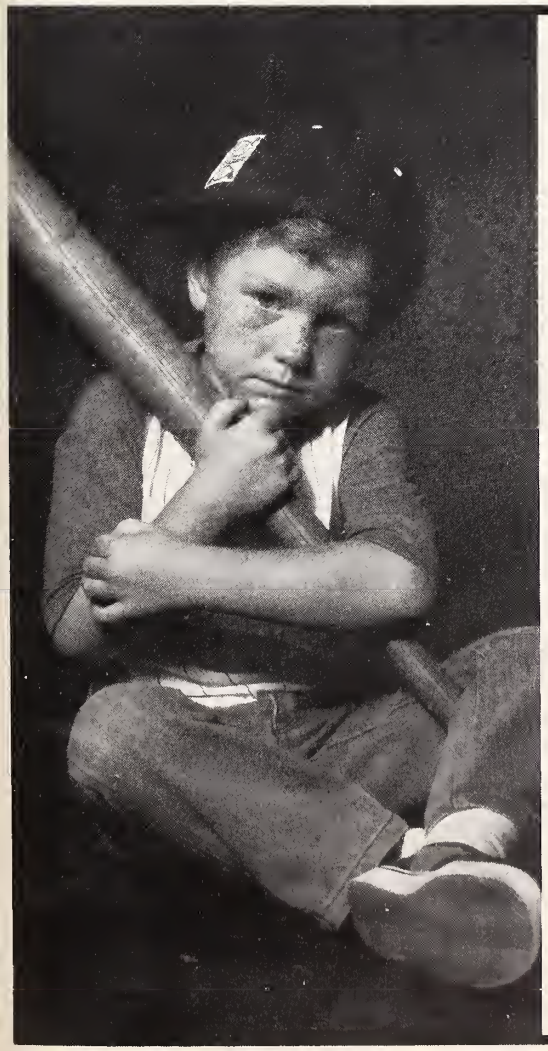
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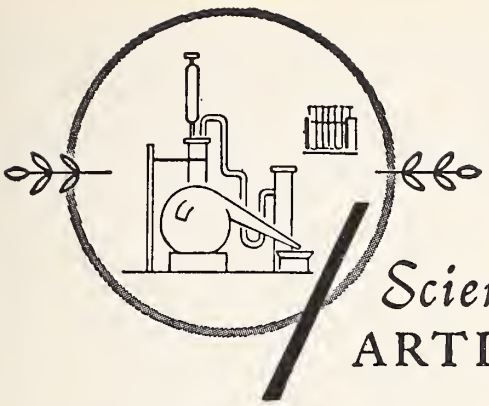
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The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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Prostatic Carcinoma

External Radiation Therapy

EASHWER K. REDDY, M.D. *and* **CARL M. MANSFIELD, M.D.,** *Kansas City, Kansas*

CARCINOMA of the prostate is the second most common malignant disease in men in the United States. It causes about 7-8 per cent of all tumor deaths in men over 50 years of age. The United States has the highest incidence of prostatic cancer.[1] For 1982, the American Cancer Society has estimated that there will be 73,000 new cases of carcinoma of the prostate in the United States, and about 23,000 patients will die as a direct result of this disease.[2] The disease is rare before the age of 35 years. The age specific incidence rates show a steady increase beyond the age of 50 years until the eigth decade. Autopsy findings in men over age 50 years, who died from other causes, revealed a 30 per cent incidence of histiological carcinomas. About 50 per cent of cancer of the prostate cases can be diagnosed by simple digital examination.

Any nodule in the prostate in a man over age 50 years is considered to be malignant until proven otherwise. It is easy to recognize these nodules on digital examination because malignant tumors of the prostate arise in the peripheral zone of the gland. By the time they become symptomatic, the tumors are locally advanced, usually extending to paraprostatic tissue and regional lymph nodes. Routine evaluation should include history and physical examination; laboratory studies such as SMA 12, CBC, and urinalysis; and tissue diagnosis obtained by perineal, transrectal, or transurethral resection of the prostate. Serum acid phosphatase with prostatic fraction, alkaline phosphatase, CT scan of the pelvis and the abdomen, and bone scan are useful in evaluating the extent of the disease. The bipedal lymphangiogram will not routinely fill the hypogastric and obturator nodes; thus it is accurate in determining the presence or absence of lymph node metastasis in about 70-75 per cent of patients.[3] Lymphatic dissemination may occur early in the clinical disease. The incidence of lymph node metastasis ranges from 27-60 per cent in Stage B and C, and increases with the size and grade of the tumor.[4, 5]

Radiation therapy is playing an important role in the curative management of prostatic cancer. A local control rate of 80-90 per cent and a five year survival rate of 60-80 per cent can be expected with adequate doses of radiation in the range of 6,000-7,000 rads in six to seven weeks. Treatment side effects and sequelae are minimal. Radiation therapy is an effective method for achieving palliation in both symptomatic bone metastases and advanced local disease.

Staging

Accurate staging is important because it determines treatment and prognosis. In 1956, Whitmore[6] proposed a staging system that separated prostatic

From the Department of Radiation Therapy, University of Kansas School of Medicine-Kansas City.

TABLE I
CARCINOMA OF THE PROSTATE

Staging			
AJCCS (TNM)	*Description*	*Whitmore*	*Jewett*
T_0	No tumor palpable; includes incidental findings of cancer in a biopsy or operative specimen.	A	A^1 (Focal) A^2 (Diffuse)
T_1	Tumor intracapsular surrounded by normal gland.		B^1 one lobe (1.5 cm or less)
T_2	Tumor confined to gland, deforming contour, and invading capsule but lateral sulci and seminal vesicles are not involved.	B	B^2> 1.5 (invading one or both lobes)
T_3	Tumor extends beyond capsule with or without involvement of lateral sulci and/or seminal vesicles.	C	C
T_4	Tumor fixed or involving neighboring structures.		
N_0	No involvement of regional lymph nodes.		
N_1	Involvement of a single regional lymph node.		
N_2	Involvement of multiple regional lymph nodes.		
N_3	Free space between primary tumor and fixed pelvic mass.		
N_4	Involvement of Juxta regional nodes.		
M_0	No distant metastases.		
M_1	Distant metastases present.	D	D

malignancy into four stages: A, B, C, and D. There are also the Jewett and TNM staging systems. All of these systems represent an attempt to predict prognosis and to better compare different treatment results. The majority of American clinicians use the Whitmore[6] or Jewett[7] classifications *(Table I)*.

Histology

Prostatic cancer is almost exclusively adenocarcinoma. Transitional cell carcinoma does arise in the large prostatic ducts, but is rare. The majority of adenocarcinomas are multicentric and diffuse. Gleason has suggested that grading of the tumors is important in predicting prognosis.[8]

Treatment

More than 50 per cent of patients have metastatic disease when the diagnosis is established. Fewer than 10 per cent of patients are candidates for curative radical prostatectomy, which carries a high morbidity with 20-30 per cent experiencing postoperative urinary incontinence and nearly 100 per cent of these patients becoming impotent. The remaining 35-40 per cent of patients present with locally advanced disease with or without the regional lymph node involvement. For these patients, radiation therapy plays an important role.

Most Stage A tumors never become clinically manifest. It has not been determined whether the transition from Stage A to Stage B or C lesion is a mere function of time or of some actual changes in the biologic behavior of the cancer cells. Patients with Stage A and well differentiated disease, who receive no therapy or conservative surgery, have a survival rate similar to the population at large.[9] The dividing of Stage A into A-1 (focal disease) and A-2 (multifocal and anaplastic) is more meaningful since the clinical behavior of Stage A-2 is more aggressive, and definitive therapy is warranted. Because the majority of patients with Stage A disease have negative nodes, only local radiation therapy to the prostate is necessary. In Stage B and C disease, 30-60 per cent of patients will have involvement of pelvic lymph nodes, and it is essential to treat the whole pelvis to include these nodes. The treatment of Stage D disease is for symptomatic relief of bone pain, as well as bleeding or obstruction from the local disease.

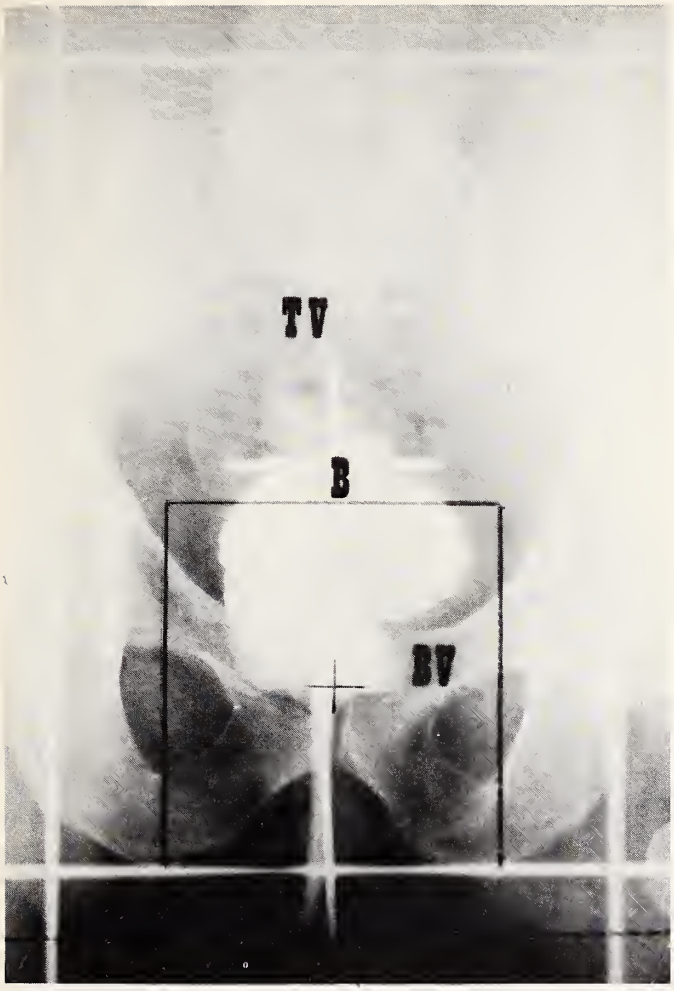


Figure 1a. Anterior simulator film showing whole pelvis and boost volumes.

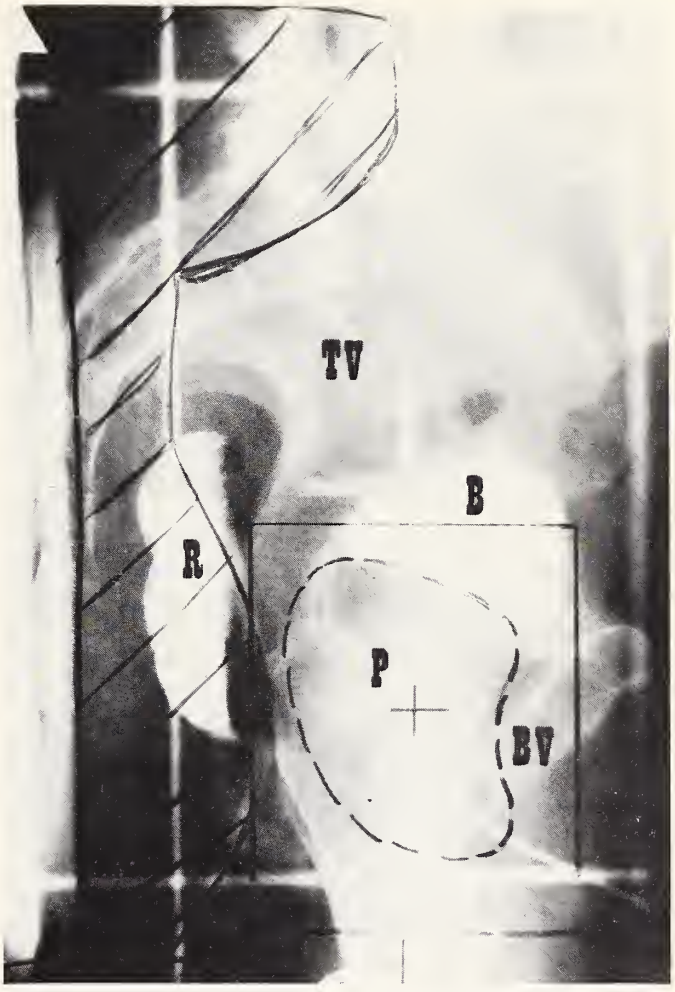


Figure 1b. Lateral simulator film with rectal and sacral block.

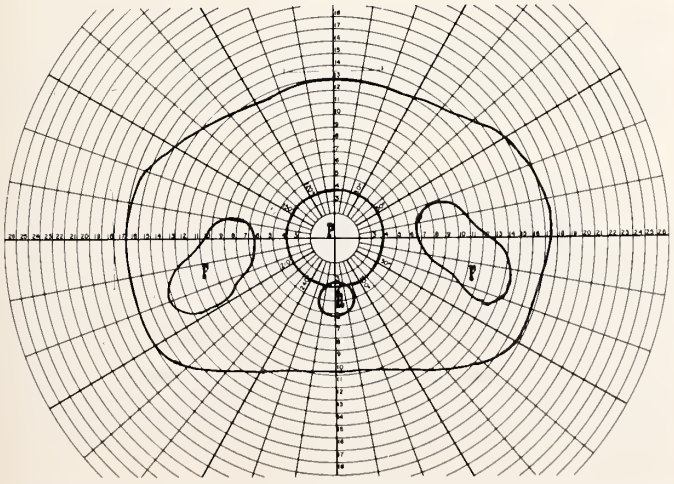


Figure 2. Patient contour taken through the center of the boost volume. P-prostate; R-rectum; F-femur.



Figure 3. CT scan taken through the center of the boost volume. P-prostate; R-rectum.

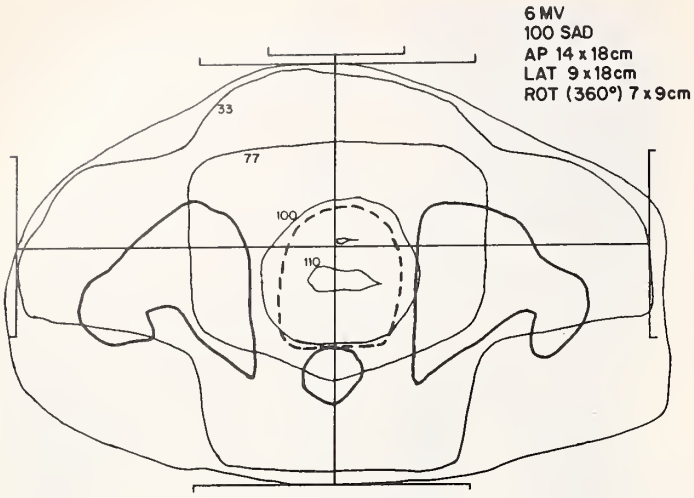


Figure 4a. Four field box technique followed by small boost field using 360 degree rotation.

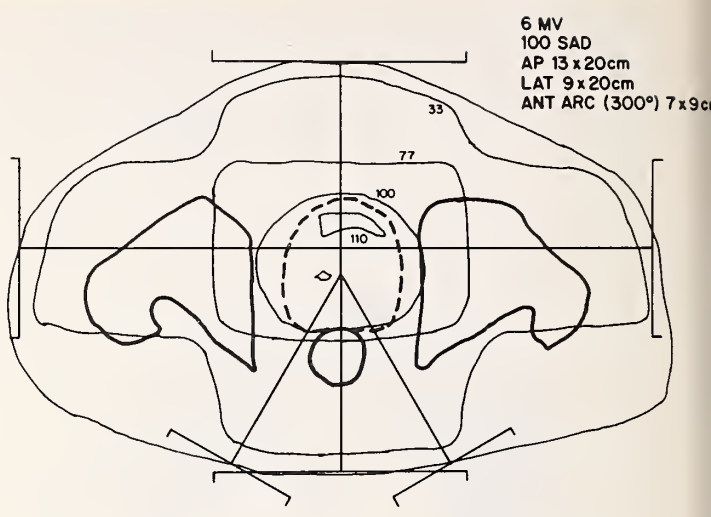


Figure 4b. Same as above but using 300 degree anterior arc for the boost volume.

Figure 4. Computer generated isodose distribution. Dotted line represents the prostate, showing different techniques to reduce the dosage to the rectum.

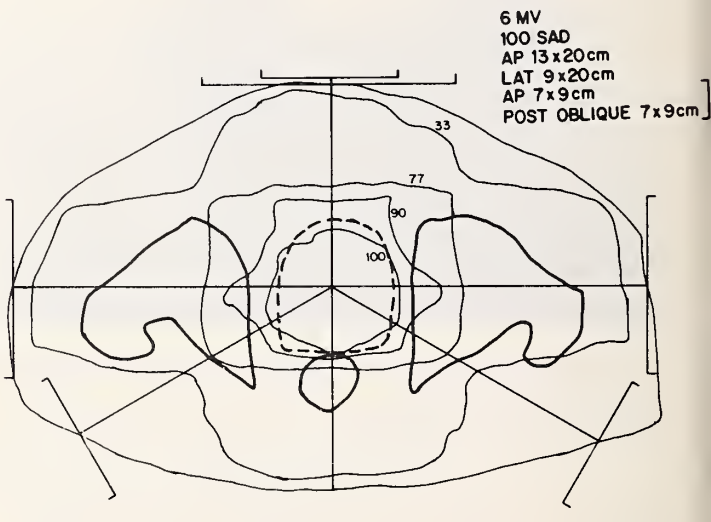


Figure 4c. Same as above but boost volume is treated using three field technique (anterior two posterior oblique fields).

Megavoltage radiation therapy units are essential to deliver a tumoricidal dosage to pelvic lymph nodes and to the prostate gland without injury to the skin and subcutaneous tissue. It is essential to use multiple field technique to minimize the dosage to vital structures, such as the rectum. The whole pelvis fields and the boost volume are marked on the anterior and lateral simulator films *(Figure 1)*. A contour is taken through the center of the boost volume, and normal structures — such as rectum and bony structures — and tumor volumes are drawn on the contour *(Figure 2)*. CT scans of the pelvis are helpful in the localization of the tumor and normal structures *(Figure 3)*. Treatment planning and isodose distribution are essential to verify the dose distribution within the treatment volume, which includes tumor and normal structures. Different combined isodose distribution for whole pelvic radiation and prostate boost are shown in *Figure 4*. Although 360 degrees rotation is common for treatment of the prostate only, other treatment techniques — such as two lateral arcs, one anterior arc, or 4 field box technique — can reduce the dosage to the rectum.

A minimum dose of 4,500-5,000 rads to the node bearing area (whole pelvis) should be delivered in 4.5-5.0 weeks at 180-200 rads/fraction at 900-1,000 rads/week. A booster dose of 1,500-2,000 rads to the prostate can be delivered immediately following completion of whole pelvic radiation, to give a total dosage of 6,000-7,000 rads to the prostate during six to seven weeks. It is recommended that all fields be treated every day in order to minimize morbidity and to obtain homogenous dosage distribution within the treatment volume.

Palliation

In carcinoma of the prostate, the most common metastatic site is bone. Symptomatic bone metastases can be effectively palliated with external radiotherapy using a moderate dosage of 3,000 rads in two weeks. When estrogen therapy is prescribed for patients with metastatic carcinoma of the prostate, the majority develop painful gynecomastia. This condition can be prevented by using a small dose of radiation to the nipple and areola of 1,200-1,500 rads in four to five fractions, prior to initiation of estrogen therapy.[10]

Side Effects

The majority of patients tolerate external radiation therapy, as a definitive course, very well, and are able to carry on their usual activities during therapy. The most common side effects are dysuria, and diarrhea which usually occurs about the third week of treatment. These symptoms can be effectively managed symptomatically with adequate oral fluids, low residue diet, and antidiarrheal drugs. After a transurethral resection of the prostate, it is essential to wait for a period of three to four weeks before initiation of radiation therapy to minimize dysuria and stricture of the urethra. If dysuria is persistant during therapy, urinalysis, culture, and sensitivity are mandatory to detect and treat any infection. Although proctitis, rectal stricture, and bowel obstruction are possible late complications, a well planned radiation treatment should result in no more than 2-5 per cent late complications. Sexual impotency following treatment of carcinoma of the prostate, using external radiation therapy, may relate not only to the specified treatment but also to the definition of the condition. The reported frequency of impotency following external radiation varies between 23 and 84 per cent. Regardless of the reported variations, at least 60 per cent of patients can maintain sexual potency following curative radiation therapy in contrast to an expected 100 per cent loss of function following radical prostatectomy.

Results

Megavoltage radiation therapy has made it feasible to deliver tumoricidal doses to the prostate without significant damage to normal tissues. A five year survival rate of 60-80 per cent[11, 12] and a local control rate of 80-90 per cent[13, 14] can be expected with external radiation therapy in prostatic carcinoma. Disease-free survival is directly related to stage, grade of the tumor, and status of the lymph nodes. Patients with positive lymph nodes have a much poorer outlook.[3]

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Pulmonary Amyloid Complications

Tracheobronchial Involvement in Primary Systemic Amyloidosis

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PADMA RAJU, M.D. *and* B. B. MUKHERJI, M.D., *Topeka*

SYSTEMIC AMYLOIDOSIS is usually viewed as a generalized disease involving multiple organ systems. In the many comprehensive reviews on the subject, lower respiratory tract involvement by amyloidosis was scarcely mentioned. During the last century, since it was first noted by Lesser[1] in an incidental autopsy finding, lung involvement by amyloidosis has been considered as a rare pathologic entity and only the subject of a few case reports or small series derived from autopsy. However, in recent years, early recognition of lung involvement by amyloid became not only possible but its pathophysiologic implications and possible importance has entered a rapid stage of evolution.

In this case report, the presence of tracheobronchial involvement in "primary" systemic amyloidosis (PSA) has been documented by fiberoptic bronchoscopy and bronchial biopsies showing the typical histologic features of amyloid. However, a transthoracic needle biopsy of an associated pulmonary nodule disclosed bronchogenic carcinoma rather than amyloid.

Case Report

A 65-year-old Caucasian male was first seen in 1977 because of dyspnea, fatigue, and palpitations. A year earlier, he had been hospitalized elsewhere for treatment of congestive heart failure manifested by biventricular cardiac enlargement, elevated venous pressure, hepatomegaly, and extensive peripheral edema. Electrocardiogram showed conduction abnormalities with right bundle branch block, first degree A-V block, and frequent premature ventricular contractions. Investigations of the underlying cause of congestive heart failure were unrevealing, and could not be explained on the basis of hypertension, valvular heart dysfunction, coronary artery disease, metabolic abnormality, or alcohol abuse. Heart failure was considered to be secondary to possible cardiomyopathy of undetermined origin. Treatment with low salt diet, diuretics, and digoxin resulted in adequate response.

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Amyloidosis of the lower respiratory tract is relatively rare but should be considered in the differential diagnosis of obstructive lesions of the tracheobronchial tree. The presence of pulmonary parenchymal nodules in primary systemic amyloidosis should not exclude the suspicion of malignancy. The association of tracheobronchial amyloidosis and bronchogenic carcinoma in the case reported here is rather unique and suggests the need for further prospective studies.

Six months later the patient developed respiratory infection accompanied by signs of consolidation of the left base. Although initial treatment with oral antibiotics had resulted in radiological clearing and some clinical improvement, he continued to complain of productive cough with scanty sputum and occasional wheezing. Treatment with bronchodilators resulted in partial relief. However, during the week prior to entry into the hospital, dyspnea became more incapacitating and was accompanied by fatigue and generalized weakness, and the patient was admitted to the hospital. Although he appeared dyspneic on minimal exertion, he denied orthopnea or paroxysmal nocturnal dyspnea.

There was no history of pleuritic chest pain or hemoptysis, but edema of the ankles had increased. There were no other symptoms suggestive of other system organ involvement. The patient had a 40-pack-year history of cigarette smoking until he had stopped smoking two years earlier. He denied history of asthma, allergy, or exposure to tuberculosis. His father died at 68 years with carcinoma of the liver, and his diabetic mother died at 70 years with cerebrovascular accident.

On admission, the patient's temperature was 37 C; pulse, 90; and respirations, 28/min. His blood



Figure 1. Radiographs of the chest (frontal projection), obtained nine months before and unchanged on entry, disclosing cardiomegaly, marked right hilar enlargement, and prominent bilateral pulmonary markings, more in the right base. Also note the left pleural thickening with calcified plaque.

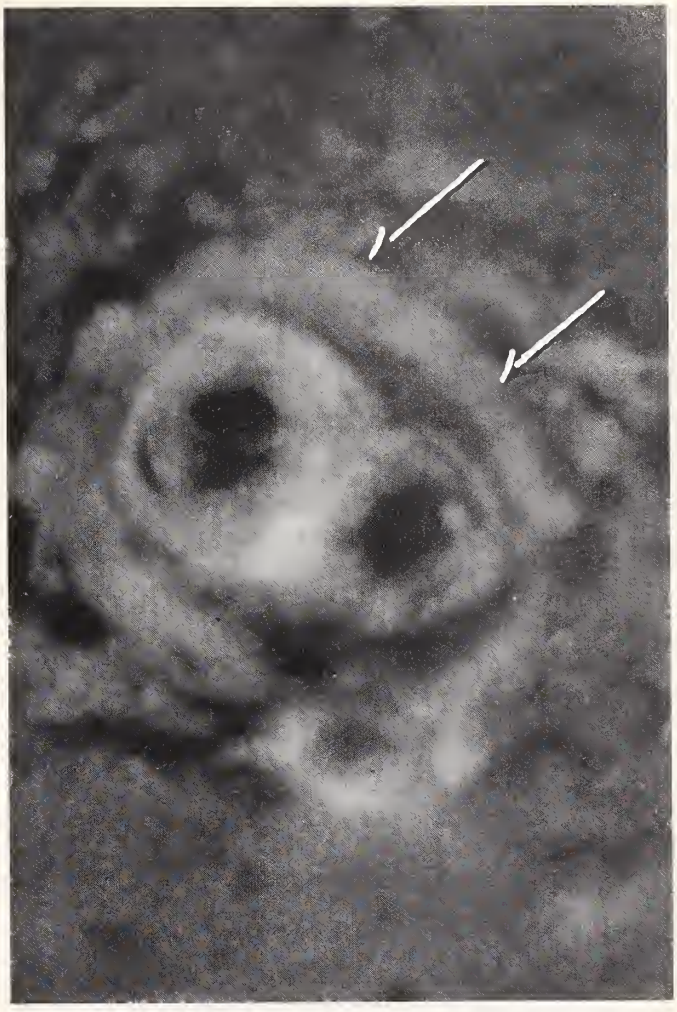


Figure 2. Apple green fluorescence of two small blood vessels of supraclavicular fat consistent with amyloid. Congo red stain viewed through polarizing microscope (750X).

pressure was 120/90 mmHg, and he was mildly dyspneic at rest. Jugular venous distension was noted which increased on inspiration but with no hepato-jugular reflux. A few small shotty cervical lymph nodes were felt, but there was no palpable thyroid enlargement. His tongue was thick with marginal indentations. Examination of the chest showed a few basal rales and diminished breath sounds in the left base with scattered rhonchi and prolonged expirations. S_3 gallop was noted, but no murmurs or precordial friction rub were heard. The liver was palpable 3 cm below the costal margin with a total span of 14 cm; the spleen was not felt. There was 1 + peripheral edema but no evidence of phlebitis, clubbing, purpuric eruptions, or cyanosis. Results of the rest of the physical examination were unremarkable.

Results of urinalysis were normal. Complete blood count showed hematocrit, 40%; WBC, 8800 with 80% PMNs; and platelet count of 215,000/cμ mm. Bleeding and clotting times were normal; sedimentation rate was five. Serum electrolytes, blood urea nitrogen, creatinine and hepatic enzymes, as well as thyroid function were normal. Chest x-ray *(Figure 1)* showed cardiomegaly with increased lung markings and evidence of right hilar node enlargement. There was a calcified plaque and pleural thickening on the left side with no evidence of free fluid on decubitus films. The EKG showed sinus rhythm with frequent PVCs and left axis deviation with incomplete right bundle branch block. In view of the presence of hilar node enlargement, cervical adenopathy and hepatomegaly, representative samples were biopsied for histological studies. Liver biopsy and bilateral scalene pad biopsy showed evidence of extensive amyloid deposition as shown in *Figure 2*. Further studies to rule out multiple myeloma or Waldenstrom macroglobulinemia were performed. This included bone marrow examination both by aspiration and biopsy; results were normal with no evidence of immature plasma cells. Bone survey

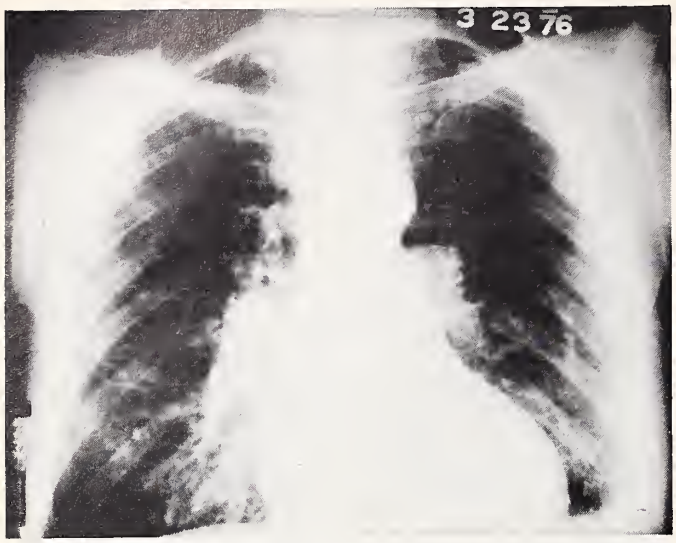


Figure 3a. Radiograph of the chest discloses a small soft tissue nodule measuring 2 x 3 cm in the left mid-lung. The heart size was normal.



Figure 3b. Tomogram of left lung showing a nodular lesion 4.0 x 2.5 cm in diameter with no calcification.

showed no lytic lesions. Studies on serum and urine protein electrophoresis and immunoelectrophoresis yielded normal results without evidence of monoclonal spike (M protein). Results of tests for Bence Jones protein were negative, and 24-hour urinary protein was less than 200 mgm. A rectal biopsy showed no evidence of amyloid deposition. An echocardiogram revealed thickening of the right and left ventricular walls with some thickening of intereventricular septum associated with hypokinesia of these walls and was consistent with infiltrative cardiomyopathy. The diagnosis of primary systemic amyloidosis was suspected with possible cardiac involvement and congestive heart failure. The patient responded adequately to low salt diet and diuretics, and was discharged on furosemide, 80 mg/day, with spironolactone, 50 mg/day; digoxin was stopped.

The patient remained well until March 1978 when he developed recurring bouts of tracheobronchitis with blood-streaked sputum. The pulmonary function tests revealed evidence of severe air flow obstruction with a moderate degree of air trapping and reduced diffusing capacity. These episodes were partially relieved by the addition of bronchodilators and intermittent oral antibiotic therapy.

In March 1979, a pulmonary nodule in the left lung was noted on chest x-ray (*Figures 3a* and *3b*) which raised the possibility of bronchogenic malignancy or parenchymal nodule due to amyloid deposit. Fiberoptic bronchoscopy was performed with local anesthesia and showed diffuse, friable, waxy, glistening tissue extending from the carina and including the mainstem and major bronchi. Almost all segmental orifices were narrowed and partially occluded by the same tissue, but segmental bronchi seen at a distance through the fiberoptic bronchoscope did not appear to be involved. Representative multiple biopsy specimens revealed submucosal, eosinophilic, amorphous hyaline material that showed green birefringence when stained with Congo red (*Figures 4a* and *4b*). The diagnosis of amyloid was further confirmed by electron microscopic examination which demonstrated the characteristic fibrillar matrix of the tissue. In view of the patient's compromised cardiopulmonary status, a transthoracic needle biopsy of the left pulmonary nodule was performed, which on histologic examination revealed squamous cell carcinoma. The procedure was tolerated well and spared the patient from a thoracotomy for the diagnosis of the associated pulmonary nodule.

Discussion

Systemic amyloidosis is rather an uncommon disease with a wide spectrum of organ involvement.

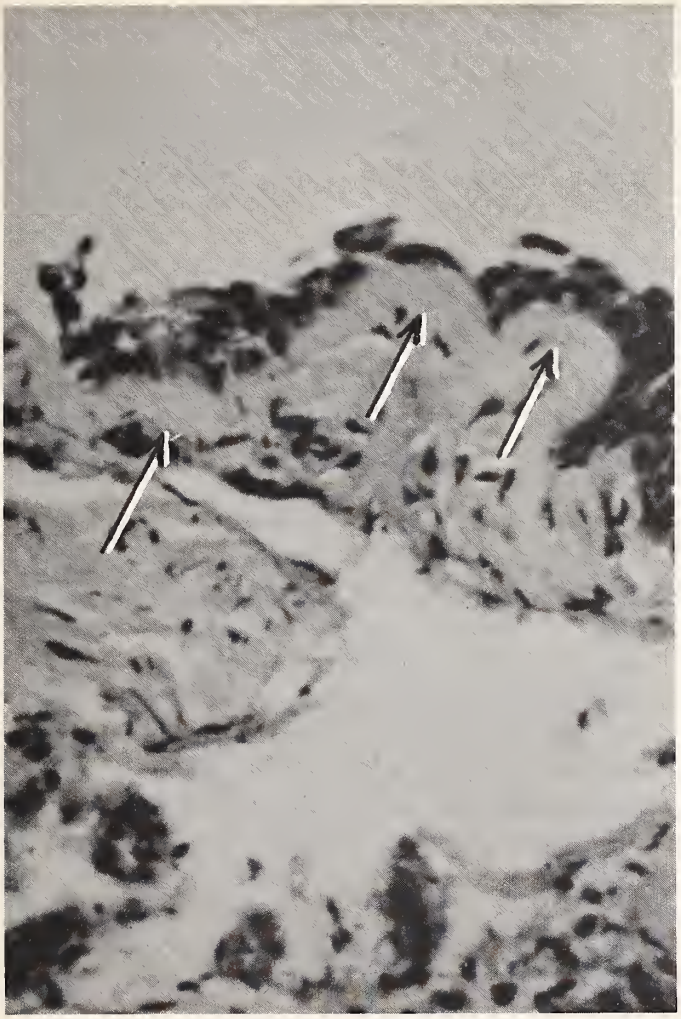


Figure 4a. Section of bronchial biopsy showing light apple green fluorescence of submucosa consistent with amyloid. Congo red view with polarizing microscope (400X).

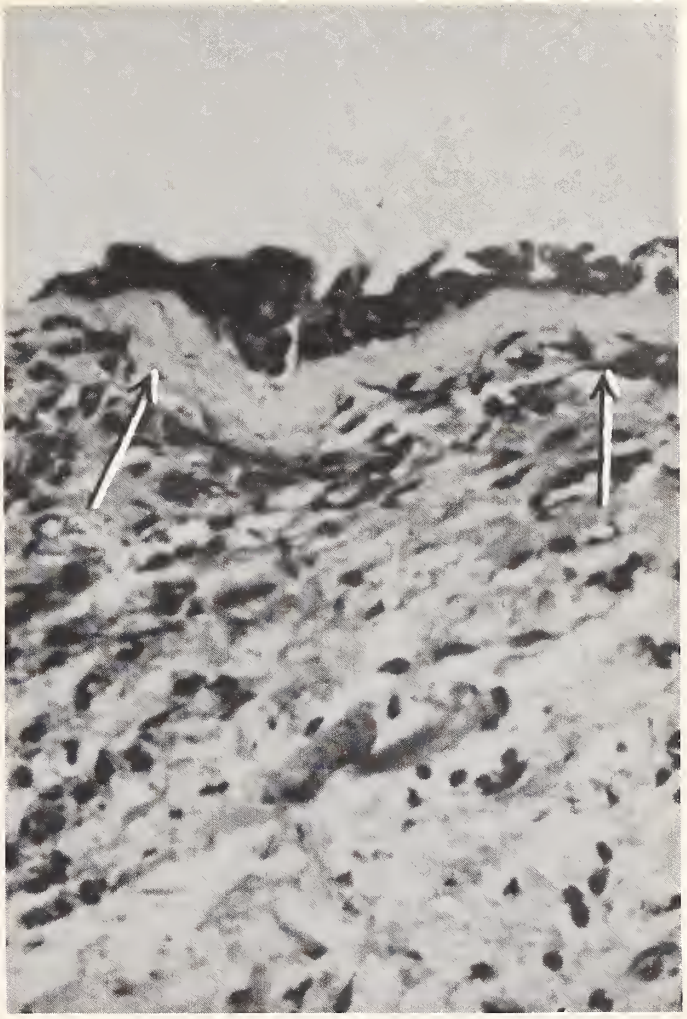


Figure 4b. Apple green fluorescence throughout submucosa consistent with amyloid. Congo red view with polarizing microscope (400X).

Although this had been recognized since the time of its discovery by Virchow[2] and was the subject of many comprehensive reviews in the literature,[3, 4] reports on lower respiratory tract involvement had been very scanty[5] and mostly autopsy derived.[6] However, in recent years pulmonary amyloidosis has been reviewed with increasing attention, not as an accidental pathological finding, but as an entity of potential clinical importance and pathophysiologic significance.[7]

Systemic amyloidosis is considered to be primary when there is no evidence of preceding or coexisting disease. In primary amyloidosis, although M component or immunoglobulin light chain may be present in the serum or urine,[8] other stigmata of multiple myeloma are absent. It also should be differentiated from secondary amyloidosis where there is underlying chronic inflammatory or infectious disease. Classification of pulmonary involvement as systemic amyloidosis by Briggs[9] and, as suggested by Prowse,[5] depicts the disease to be of two major varieties — tracheobronchial and parenchymal — and if parenchymal, either nodular (single or multiple) or diffuse. Based on histologic studies, the lung was frequently involved in primary systemic amyloidosis with incidence ranging from 36 per cent[10] to 90 per cent.[6] Cilli *et al.*,[7] through a careful clinical and pathological correlative study, found 92 per cent of patients with primary amyloidosis had pulmonary amyloid deposits, and symptoms were attributable to these deposits in 40 per cent of cases.

Tracheobronchial amyloidosis is a relatively frequent form of amyloidosis of the lower respiratory tract.[11] The average age of patients in this form of amyloidosis is 53 years with a male/female ratio

(Continued on page 510)

Viral Hemorrhagic Fevers

Medico/Geographic Ramifications

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VIRAL HEMORRHAGIC fevers were first classified together by D. C. Gajdusek in 1962 as a group of human diseases characterized by fever, severe hemorrhagic state, and fearsome pathogenicity. The causative agents of most of these fevers have been identified as members of three different RNA virus families; namely, Arenaviridae, Bunyaviridae, and Togaviridae. However, the pathogenesis and pathology of these diseases have not yet been unequivocally delineated. Moreover, these fevers differ from one another in geographic distribution, ecology, mode of transmission, and route of infection. The Argentine, Bolivian, and dengue hemorrhagic fevers — as well as yellow fever — occur in the Americas; the rest are encountered in Asia, Europe, and Africa. As no vaccine or specific treatment is currently available for these diseases, they, therefore, cause significant health problems for many countries throughout the world.[1, 2]

Viral hemorrhagic fevers are severe, often fatal diseases with similar clinical features but of diverse etiology. The majority have been recognized during the last 30 years. They occur mainly in Asia, Africa and South America, and hence are considered exotic in this country. The increase in international travel could result in importation and subsequent community or hospital outbreaks of these afflictions.

Classification

About a dozen diseases are now included in this group. The etiological agents of all but two have been classified *(Table I)*.

As the names of the diseases indicate, these infections are of worldwide distribution. All have hemorrhagic features of complex and little-understood pathogenicity, and are mostly severe and often fatal. They have been placed in four categories: (1) tick-borne: Crimean-Congo and Omsk hemorrhagic fevers and Kyasanur Forest disease; (2) mosquito-borne: dengue hemorrhagic fever and yellow fever; (3) zoonotic: Korean, Argentine, and Bolivian hemorrhagic fevers and Lassa fever; and (4) African hemorrhagic fever: Marburg and Ebola fevers. A concise description of the important features of these diseases follows.

Arenavirus Hemorrhagic Fevers. The Argentine disease (caused by Junin virus isolated in 1958 from a patient's blood in Junin), which was recognized in the early 1950s, is an occupational disease among farmers in the corn fields of Argentina at harvest time when the rodent population is at its peak. The disease has an acute febrile period followed by renal, hematological, neurological, and cardiovascular changes. The more severe but less common forms are associated with hemorrhage, convulsion, profuse bleeding, and shock. Incidence varies with climate and other ecological factors; however, several hundred cases are reported each year. The case fatality rate is 10-20 per cent. Recently, an immunofluorescent test on urinary sediment cells for the diagnosis of this disease as early as 24-48 hours after onset of symptoms was described.[3] The Bolivian disease (caused by Machupo virus isolated in 1963 from a patient's spleen) resembles the Argentine disease with malaise, fever, headache and myalgia, and has a similar case mortality rate. However, its incidence has decreased drastically in recent years due to rodent control. The above two arenavirus diseases have not been observed outside of their endemic areas. The third hemorrhagic fever in this group — *i.e.*, Lassa fever — was first recognized in January 1969 in an American missionary nurse in Lassa, Nigeria. The patient was transferred to a larger hospital in Jos, where the two missionary nurses who attended her contracted the disease in February. The index case and one of the two nurses died; the other nurse was aeromedically flown to New York City where she was treated and subsequently recovered. In December 1969, a Lassa fever patient was admitted to the Jos hospital resulting in a hospital-centered outbreak involving 27 cases (mostly secondary but a few tertiary). Thirteen deaths (including a resident physician) occurred dur-

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TABLE I
CLASSIFICATION OF ETIOLOGICAL AGENTS IN VIRAL HEMORRHAGIC FEVERS

Etiological Agent	*Disease*
Family	
Arenaviridae	Argentine hemorrhagic fever, Bolivian hemorrhagic fever, and Lassa fever.
Bunyaviridae	Crimean-Congo hemorrhagic fever, Korean hemorrhagic fever, and Nephropathia epidemica.
Togaviridae	Dengue hemorrhagic fever, Omsk hemorrhagic fever, and Kyasanur Forest disease (the yellow fever virus also belongs to this family).
Unclassified (African hemorrhagic fevers)	Marburg fever and Ebola fever.

ing January and early February 1970. Subsequently a number of outbreaks involving several hundred cases — some hospital-associated (human-to-human), and others acquired at home or in other community surroundings (non-human cycle of transmission) — have occurred in Nigeria, Sierra Leone, and Liberia. Moreover, serological studies indicate the presence of this virus in other countries of west, central, and east Africa. The causative virus was isolated in March 1969 at Yale University School of Medicine from blood serum and thoracentesis fluids obtained from the first three cases. The agent is highly pathogenic and hazardous; it has caused laboratory-acquired infections, one of which ended fatally (at Yale University). A second infection at Yale University occurred in the internationally renowned arbovirologist, Dr. Jordi Casals, who worked with the virus. He was successfully treated with serum obtained from the recovered missionary nurse. The virus resists a temperature of 56C for 30 minutes but is inactivated at 60C for 60 minutes.[4] The disease in man ranges from mild, acute febrile illness of short duration to a prolonged fatal infection involving multiple organs with severe toxemia and manifestations of pharyngitis, pneumonitis, myositis, myocarditis, nephropathy, and hemorrhage. Mortality in hospitalized cases ranges from 35-50 per cent. However, a recent study of this disease in Sierra Leone indicated a relatively mild course among children as compared to adults; the most prominent features in these children were fever, hepatosplenomegaly, vomiting, seizures, abdominal pain, cough, and malaise.[5]

The agents of these three diseases are mai in nature by means of persistent or long-term tions of several species of rodents. The Junin virus is associated with at least three species of cricetine rodents including Calomys with which the Machupo virus is also associated. The Lassa fever virus is associated with Mastomys natalensis. These rodents excrete virus in the urine and in secretions of nose and mouth and these, in turn, can contaminate food, drink, and dust from which humans may acquire infection. Moreover, inoculation of infected rodent excreta through skin abrasions may also be involved in human infection. Rat control has proved an effective prophylactic measure at least in Argentine disease, and early administration of immune serum to patients with these diseases — especially Lassa fever — has had good results. In March 1976, an ill 42-year-old Peace Corps woman arrived in Washington, D.C. from Sierra Leone via London after travelling on two different aircrafts. Her illness was soon diagnosed when Lassa virus was isolated from her urine. However, she recovered and the disease was not contracted by her 300 contacts.

Bunyavirus Hemorrhagic Fevers. Two categories — tick-borne Crimean-Congo hemorrhagic fever, and zoonotic Korean hemorrhagic fever and nephropathia epidemica — are caused by bunyaviruses.

1. The Crimean syndrome was first recognized in Western Crimea in 1944 and its agent was isolated from patients' blood and ticks in 1967. The Congo syndrome was first recognized in Zaire (formerly Belgian Congo) and its agent was isolated from the blood of a febrile child in 1956. The close antigenic and biologic relationship between the two agents was reported in 1969; hence the designation Crimean-Congo hemorrhagic fever was introduced. The disease is now known to occur in large areas around the Black and Caspian Seas, in Bulgaria, Yugoslavia, Greece, East and West Africa, South Africa, Iraq, United Arab Emirates, Pakistan, and possibly elsewhere. Moreover, South Africa had its first indigenous case of this disease on February 14, 1981. The patient was a 13-year-old boy who had attended camp and from whose scalp a Hyalomma tick had been removed. He died on February 19 in a private hospital despite vigorous treatment.[6]

The virus is maintained in nature by asymptomatic infection in the ticks of Hyalomma genus (transavarian transmission) and a variety of domestic and wild animals; *e.g.*, sheep, cattle, goats and hares (tick-bite transmission). Agricultural workers are at the highest risk especially during spring and summer when ticks are most active. Moreover, transmission

via blood or bloody discharges from patients has been responsible for numerous hospital-associated cases (including several attending physicians and nurses) reported recently from Pakistan, Iraq, and Dubai.[7, 8]

The disease has an incubation period of three to six days and starts abruptly with fever, chills, headache, myalgia, abdominal pain, and vomiting. Hematemesis, melena, and wide-spread hemorrhagic manifestations ending in hypotension and shock may follow. Histologically, extensive cellular necrosis and hemorrhage in the liver, necrosis and lymphoid depletion in the spleen, congestion and edema in the lungs, and hemorrhage in the kidneys, adrenals, and intestine are seen. The mortality rate ranges from 10-50 per cent. A vaccine is available in the U.S.S.R.

2. The Korean hemorrhagic fever was first recognized in 1951 when United Nations personnel (including American troops) contracted the disease during the Korean war. The disease is known by many names and is found in a geographical area, extending from Japan to Europe, which is inhabited by one-half of the world's population. Its agent (now called Hantaan virus) was identified in 1976 in the striped field rodent Apodemus agrarius coreae by the immunofluorescence technique (using patients' acute and convalescent sera as antibody and infected rodent lungs as antigen). Subsequently the virus was isolated in cell cultures (A-549 cells derived from a human lung carcinoma) in 1979 and characterized as a bunyavirus in 1981.[9] The etiological link between the Korean and other Far-Eastern (Far-Eastern U.S.S.R., Japan, and People's Republic of China) syndromes and a clinical syndrome termed nephropathia epidemica in Scandinavia was reported in 1979 when the same immunofluorescence technique, mentioned above, was used with patients' sera and the red-backed bank vole, Clethrionomys glareolus; however, the two etiological agents are not identical.[10]

Both the Far-Eastern (Korean, etc.) and the Scandinavian illnesses have renal syndrome. However, nephropathia epidemica is clinically much milder with fewer hemorrhagic manifestations and a much lower mortality rate. The Far-Eastern disease has a sudden onset of chills, fever, lethargy, headache, myalgia, costovertebral pain, suffusion of face and upper thorax, petechiae or large skin hemorrhages, as well as a pronounced renal involvement (renal tubular lesions) with proteinuria and oliguria which may lead to anuria. Death, which occurs in 1-2 to 25-30 per cent of cases (the higher rate seen in the Far East), is associated with shock. Nephropathia epidemica, on the other hand, is a disease in which the clinical picture is dominated by an acute tubular and interstitial nephritis. The disease starts abruptly with fever, headache, and malaise followed on the fourth day by vomiting, facial flush, back and abdominal pains, and the start of the renal syndrome (albuminuria, oliguria, and sometimes anuria). The hemorrhagic symptoms, which occur in up to 10 per cent of cases, consist of petechiae in the palatal arches and soft palate, nasal hemorrhage, and melena. The mortality rate is about 1 per cent.[11]

Togavirus Hemorrhagic Fevers. This group includes dengue and Omsk hemorrhagic fevers and Kyasanur Forest disease. Yellow fever, which still occurs sporadically in rural areas of West Africa and South America is also included in this group. The main disease in this group is dengue which has been extensively studied in recent years. As regards Omsk hemorrhagic fever, it is restricted to Omsk and Novosibirsk Oblasts in Siberia where the disease is transmitted to humans either by tick bite or by direct contact with infected muskrats which are used for hunting purposes. The case fatality is 0.5-3.0 per cent. Kyasanur Forest disease is restricted to Mysore State in India. The second phase of this disease shows hemorrhagic manifestations of epistaxis, hematemesis, hemoptysis, melena, bleeding gums, and occasionally bleeding uterus; however, the mortality rate is low. Infected ticks transmit the clinical disease to forest-dwelling monkeys and the villagers in the Kyasanur Forest area.

Dengue infection occurs in the tropical regions throughout the world involving a population of 1.5 billion inhabiting the Pacific islands, Indonesia, the Philippines, Malasia, Singapore, Vietnam, Thailand, Burma, eastern India, Sri Lanka, West Africa, and the Caribbean region. However, in 1980, after an absence of 40 years, dengue virus (serotype 1) circulated within the continental United States (Texas). As its vector, Aedes aegypti, is prevalent in Texas, Louisiana, Mississippi, Alabama, Georgia, Florida, North and South Carolina, Tennessee and Arkansas, the extent of the current virus circulation in this country and whether North Americans are now at risk to dengue hemorrhagic fever, are undetermined. The etiologic virus has four serotypes and is transmitted to humans mainly by four species of Aedes; *i.e.*, aegypti (the most important), albopictus, polynesiensis, and scutellaris. The disease caused by this agent is of two forms: classical dengue fever and dengue hemorrhagic fever.

The classical form (breakbone fever) occurs mostly in adults and older children; it starts abruptly with fever (often biphasic), headache, pain behind the

eyes, chilliness, and general severe myalgia. A maculopapular rash generally appears on the trunk which later spreads to the face and extremities. The disease is much milder and the upper respiratory symptoms are prominent in younger children. It generally lasts for about ten days ending usually with complete recovery.

Two U.S. travelers (residents of Illinois and Virginia) developed dengue serotype 4 infection in April 1981, after returning home from St. Barthelemy Island (French West Indies). Dengue serotypes 2 and 3 have been present in the Caribbean region at least since the 1960s and dengue serotype 1 was first recognized in Jamaica in 1977. However, the above two cases represent the first importation of serotype 4 to the Western Hemisphere.

Dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS) has recently become a considerable growing public health problem in Southeast Asia, India, and the Pacific islands (12,000 deaths among 350,000 hospitalized patients), and is currently providing a very rich field for research. It is still unknown why the dengue virus has, in recent years, caused a rather mild disease in one human host but a severe, often fatal, disease in another. The hemorrhagic form, however, occurs mostly in children aged 2-13 years. It was first recognized in 1954 in the Philippines and later in other Southeast Asian countries and was linked with dengue virus in 1956. Seroepidemiological studies have indicated that DHF/DSS occurs in individuals who have had a previous heterologous dengue fever infection or who had acquired heterologous maternal dengue antibodies. It has two stages, the first starting abruptly with fever accompanied with dengue-like symptoms described above. However, as the temperature falls, the condition of the patient suddenly deteriorates with hypotension, peripheral vascular congestion, and petechiae. The patient is restless and sweating and has cold clammy extremities and a hot feverish trunk. Moreover, shock occurs in different degrees in these patients. If not treated, death may ensue in a few hours. The critical period in the disease, however, is the fourth and fifth days and the occurrence of purpura, ecchymoses, epistaxis, hematemesis, melena, convulsions, and severe shock during these two days indicate a poor prognosis. However, if a patient survives this period, recovery is usually complete. While death is rare in the classical dengue fever, the mortality rate in DHF/DSS is 5-10 per cent. Moreover, as the factors involved in the pathogenesis of this syndrome have not yet been delineated, the potential for worldwide occurrence of DHF/DSS now looms in the background. In this connection, the first case of non-fatal dengue hemorrhagic fever in North America was reported in 1980 in a 22-month-old Loatian male infant who arrived in Canada from a Thai refugee camp. The child developed typical symptoms including disseminated intravascular coagulation with bleeding into the skin, gastrointestinal and genitourinary tracts shortly after arrival. Serological tests confirmed the clinical diagnosis.[12] A live attenuated dengue vaccine is now being developed in Thialand (Bangkok) and the U.S. (Hawaii) under the auspices of the WHO.

African Fevers Caused by Unclassified Viruses. Two clinically very similar and severe hemorrhagic febrile illnesses apparently originating in Africa named Marburg (West Germany) and Ebola (a river in Northern Zaire) fevers are included in this group. They are caused by two morphologically identical but antigenically distinct viruses which, unlike other known animal viruses, have elongated, filamentous, tubular with branching or other bizarre configurations.

Marburg fever was first recognized in August 1967 among laboratory workers in Marburg and Frankfurt (West Germany) and Belgrade (Yugoslavia) who had direct contact with blood, organs, or cell cultures derived from African green monkeys imported from Uganda. A total of 31 cases (several of them secondary, including cases in hospital personnel by contact with patients' blood) with seven deaths (mortality rate: 29%) occurred between August and November 1967 (the last case acquired venereally by a woman from the infected semen of her recovered husband). Subsequently, in February 1975, a 20-year-old Australian student hitchhiking with a 19-year-old female student companion through Southern Rhodesia fell sick there and died of Marburg fever in a Johannesburg hospital. Shortly after, his companion and a 20-year-old nurse who cared for him, developed the disease; however, both women recovered. The third outbreak of this disease occurred in January 1980, in Kenya. A French electrical engineer working in a sugar factory in Western Province developed Marburg fever and died seven days later at the Nairobi Hospital. A physician who attempted resuscitation of this patient developed the disease nine days later and was admitted to the same hospital. However, he recovered, and because of a containment program initiated at that hospital, no nosocomial transmission occurred. The virus was isolated from the semen of this patient two months after his clinical recovery.[13]

Ebola fever made its appearance between July and November of 1976 as severe outbreaks in Northern Zaire and Southern Sudan. In Zaire, there were 237

cases with 211 deaths (mortality rate: 89%); in two cities in Sudan, there were 299 cases with 150 deaths (mortality rate: 50%). In a hospital in Sudan, where Ebola patients were admitted, 76 of the staff developed the disease and 41 of them died.[14]

Marburg and Ebola fevers, which are virtually indistinguishable, start with severe headache followed by high fever and generalized myalgia, especially in the back. Prostration and watery diarrhea leading to rapid weight loss and dehydration soon follow. Nausea, vomiting, and abdominal pain persist for several days. A maculopapular rash (less obvious on black skins), followed by desquamation and a very dry throat, are also present in most cases. In the majority of both Marburg and Ebola cases, severe bleeding — most frequently involving the gastrointestinal tract and lungs — with hematemesis and melena are observed. Moreover, bleeding from the nose, gums, and vagina as well as subconjunctival hemorrhage are common. Death is generally preceded by severe blood loss and shock.

Although the agent of Marburg fever was imported to Europe by African monkeys, these animals are not the natural reservoirs of this infection. Likewise, the source of Ebola fever outbreaks in Africa is still unknown. However, in both fevers, man-to-man transmission by close and prolonged contact (especially with patient's blood) is well established.

Pathogenesis

The pathogenesis of viral hemorrhagic fevers is currently the least understood aspect of these diseases; a number of hypotheses for explanation of its mechanism have recently been proposed. Among all of these diseases, the dengue hemorrhagic fever is the most perplexing and also the most extensively studied. This is because firstly, the dengue virus is currently causing a mild disease in one individual and a severe, often fatal disease in another, and secondly, dengue infections are now worldwide in distribution, occurring in more than 60 countries.

As the taxonomic diversity of the etiologic agents of these diseases was elucidated, the old concept that hemorrhagic fevers have the same clinical and pathologic features was abandoned. In 1967, disseminated intravascular coagulation (DIC) was proposed as the cause of bleeding in all hemorrhagic fevers as this phenomenon was observed in monkey yellow fever, Korean hemorrhagic fever and, to some extent, in dengue hemorrhagic fever. However, DIC is not evident in nephropathia epidemica, and consumption of clotting proteins has not been demonstrated by ante- or postmortem examination of Argentine hemorrhagic fever cases. Thus, DIC is not currently considered a common mechanism in these diseases.

When it was shown that DHF/DSS occurs in individuals infected with a second heterotypic dengue virus, the concept of immunopathogenesis as the underlying mechanism in this disease was proposed. Indeed, immune complexes are seen in the kidneys of DHF/DSS patients as well as in the kidneys of patients with certain primary (first infection) hemorrhagic fevers with renal syndrome (*i.e.* Korean hemorrhagic fever and nephropathia epidemica). In other hemorrhagic fevers (*i.e.* Argentine disease), however, no evidence of immunopathogenesis is observed. Furthermore, the nature of the depositions, the delayed appearance of circulating immune complexes and the unchanged complement level in these patients indicate a secondary rather than primary role for immune complexes. Here again, immunopathogenesis does not appear to be a common mechanism among hemorrhagic fevers.

A more recent approach to understanding the pathogenetic mechanisms in hemorrhagic fevers has focused on the growth of the causative viruses in cells of mononuclear phagocytic lineage, *i.e.*, the reticuloendothelial system (RES). This approach has been vigorously pursued with Argentine and dengue hemorrhagic fevers which are caused by an arenavirus (Junin) and a togavirus (dengue) respectively. A remarkable similarity in sites of virus replication for both diseases has been observed; both Junin and dengue viruses grow in the cells of RES and cause their destruction. Moreover, the mononuclear macrophages are also commonly the sites of replication for the viruses causing other hemorrhagic fevers. These cells have abundant specific receptors for arenaviruses which facilitate their infection. However, RES cells have few receptors for dengue virus. Here, it is believed that the heterotypic antibodies from a previous dengue infection play the unusual role of virus-specific receptor and thereby the entry of the virus into the RES cells is facilitated. Hence the new hypothesis of immune infection enhancement based on the central concept of antibody-mediated infection of RES cells by dengue virus has been proposed for DHF/DSS. This hypothesis conceives that initially dengue-specific immunocompetent cells (possibly T lymphocytes), present in the host from a previous dengue infection, activate the patient's dengue-infected mononuclear phagocytes, causing the release of products known to interact with the complement and blood clotting systems. In addition, as these cells also release a vascular permeability factor, all signs and symptoms of DHF/

DSS could be attributed to the effector activities of RES.

This mechanism can underlie the common pathologic features (*i.e.* vascular permeability, complement activation, and DIC) of various viral hemorrhagic fevers. However, DHF/DSS appears unique in requiring heterologous or group-specific antibodies (as cell receptors) for the infection of RES cells with the causative virus. Studies with dengue monoclonal antibodies have indicated that some possess strong neutralizing activity but no enhancing activity (serotype-specific or homologous), and others possess both strong hemagglutination inhibition and enhancing activities (group-specific or heterologous). Halstead has proposed that infection of mononuclear phagocytes with dengue virus is a kind of biologic Yin-Yang phenomenon. Here the opposing forces of neutralizing and enhancing antibodies are leavened by small portions of the opposite principle. Thus the outcome of dengue virus infection in humans is either severe due to the prevalence of enhancing (Yin) antibodies or conversely mild due to the prevalence of neutralizing (Yang) antibodies. Two other known factors are also related to risk to DHF/DSS. They are: sex-age dependence, *i.e.* females at and after the age of 4 years have nearly twice the risk of developing shock syndrome as compared to males of the same age group; and good protein-calorie nutrition seems to have an adverse effect on the outcome of this disease. In this connection, it should be pointed out that females and well-nourished individuals are generally considered more immunologically competent than males and malnourished ones. Moreover, genetic factor(s) may also be involved.

Once the cells of RES are infected with the agents of these different hemorrhagic fevers, the consequence of events leading to hemorrhagic manifestations is believed to be similar in all these diseases. The known pathogenetic elements produced by enzymes released from the activated mononuclear phagocytes are the cleavage of C3 into C3a and C3b, the release of tissue thromboplastin, and generation of vascular permeability factors. These effector phenomena can account for all the major manifestations of hemorrhage and shock seen in these diseases.[15-18]

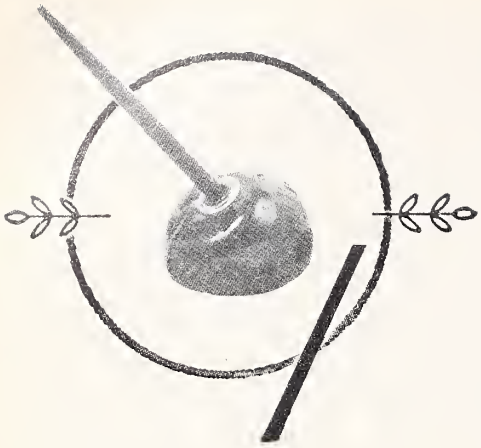
Discussion

Hemorrhagic manifestations in two viral diseases — *i.e.* smallpox and yellow fever — have been known for many centuries. However, most of the hemorrhagic fevers reviewed here have come to light within the last 30 years. Currently many questions about these diseases remain unanswered. Are they new or newly discovered? Will there be additional hemorrhagic fevers appearing in the future? Will those currently restricted to certain geographical areas remain confined to their endemic areas? Should we in the United States be concerned about these exotic diseases?

These so-called new hemorrhagic fevers probably existed in nature as silent enzootic foci for many years; however, it is believed that in the past only few individuals in endemic areas were accidentally exposed and infected. Hence these diseases have been referred to as old diseases which have recently become prominent with new names. As humans are constantly exploring new areas, domesticating animals and developing agriculture, there probably will be additional exposure to certain hitherto unrecognized enzootic infections which will appear as new human diseases. In regard to our concern about these diseases, firstly, the well-being of our fellow-humans anywhere should be our concern, and secondly these exotic diseases of distant lands may not remain exotic forever as they can be brought into the United States at any time by the ever-increasing American international travellers. This is particularly dangerous in a number of these diseases (*e.g.* Lassa fever, Crimean-Congo hemorrhagic fever) in which after the transmission of the disease to humans from the original reservoir host, person-to-person transmission, in a community or in a hospital, has been extensive. A recent documented case of importation of Lassa fever by an American, who contracted the disease in Africa, into Washington, D.C. is a vivid example. Four other importations of this disease to Europe (three to England and one to West Germany) occurred between 1972 and 1976. Moreover, infected monkeys from Africa have caused severe outbreaks of Marburg fever in Europe, and clinical specimens from Lassa patients in Africa have caused infection in two laboratory workers at Yale University (one of which ended in death) and, more recently, two others at CDC. Hence, for practical reasons, these exotic fevers — wherever they occur in the world — are important, and physicians, nurses, laboratory technicians, and public health personnel should be alerted to their potential danger.

The risk of importation of these exotic diseases to the United States is now considered high; indeed because of increasing travel by Americans to developing and tropical countries, it seems almost inevitable. Hence, U.S., Canadian, and a number of European governments have recommended certain measures for the containment of any importation.

(Continued on page 496)



Clinical NOTES

A New Perspective

Blood Pressure in Feet and Inches of Blood

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ENGLISH PHYSIOLOGIST and divinity student Stephen Hales is credited as being the first to measure blood pressure (1733). His subject was a horse. He used a glass tube which had its origin in the carotid artery. The blood was forced straight up a glass tube for a distance of more than eight feet.

To help patients to better understand their blood pressure, a visual method was used recently on nurses and paramedical help at the Atchison Hospital *(Figure 1)*. It consists of a blood pressure unit with a Tycos or mercury manometer, a flask containing two ounces of colored saline solution of known specific gravity (in the range of normal blood), and an upright board carrying clear plastic tubing up to the ceiling along a tape measure 99 inches long. The flask containing the colored saline is sealed with a two-holed rubber stopper. The floor-to-ceiling plastic tubing leads through the first hole via glass tubing to the base of the flask containing the colored saline solution (specific gravity 1.066). A short glass tube through the other stopper hole communicates with a blood pressure bulb, a Tycos manometer, and an arm pressure cuff on the arm of the patient. The Tycos and the feet and inches measurement of saline (as a blood substitute) may be read simultaneously.

Todd and Sanford[1] and Winthrobe[2] give the specific gravity of normal range of human blood as 1.048-1.066, dependent largely upon the amount of hemoglobin it carries; red cells have a specific gravity of 1.092-1.095. The diurnal variation is about .003. Winthrobe[2] further states that the pycnometric method was used to establish the limits of specific gravity of blood. This is generally lower in the afternoon and after meals, and higher after exercise and during the night. Blood with its full quota of hemoglobin has a specific gravity of about 1.066. Therefore, it is within reason to accept a specific gravity of 1.06663 as a standard. In addition, this specific gravity makes our mathematical solution more nearly correct, as 24 millimeters of mercury and one foot of blood of a specific gravity of 1.06663 both have the same relative weight of 325.1088.

A solution with a specific gravity of 1.066 may be made by dissolving 98 gm of sodium chloride/liter of water at 20 C.

A series of blood pressure readings revealed the surprisingly precise similarity between one foot of saline solution and 24 mm mercury; *i.e.*, one foot (12″) of saline solution with a specific gravity of 1.06663 equals, in relative weight, 24 mm mercury. Therefore, one-half the number of millimeters of mercury is equal to the number of inches of blood.

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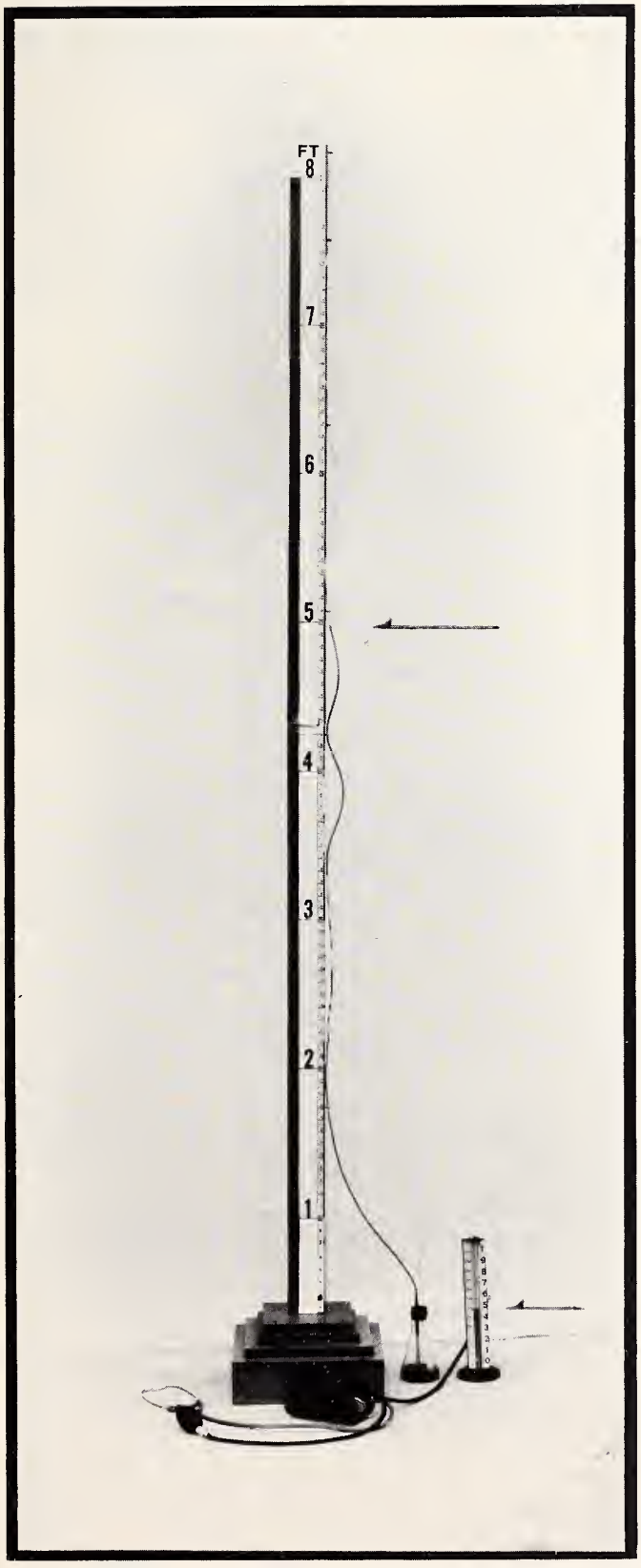


Figure 1. A measurement of the blood pressure shown both in mercury and in saline solution with a specific gravity of 1.066. The saline has been colored black with India ink. Arrows designate the levels of blackened saline and of mercury.

To aid in the conversion of a column of mercury into a column of blood, a strip of white one-half inch adhesive tape may be placed along the right border margin of the manometer *(Figure 2)*. This strip is divided into segments 24 millimeters long to denote feet of blood and subdivided into two millimeter segments to designate the inch-markings using the usual manometer scale.

The scale on the mercury manometer is about 5 per cent less than on the metric ruler. Therefore, the manometer scale should be used because it measures the distance from the surface of the mercury in the reservoir cup to the height of the observed mercury column. As the mercury in the observed column rises, the level in the reservoir cup falls; therefore, the adjusted measurements. The ordinary mercury manometer shows the 300 mm to be less than 290 mm on the metric scale.

A patient with a blood pressure of 120/72 may also be told that his/her blood pressure in blood is 60 inches over 36 inches of blood, or five feet over three

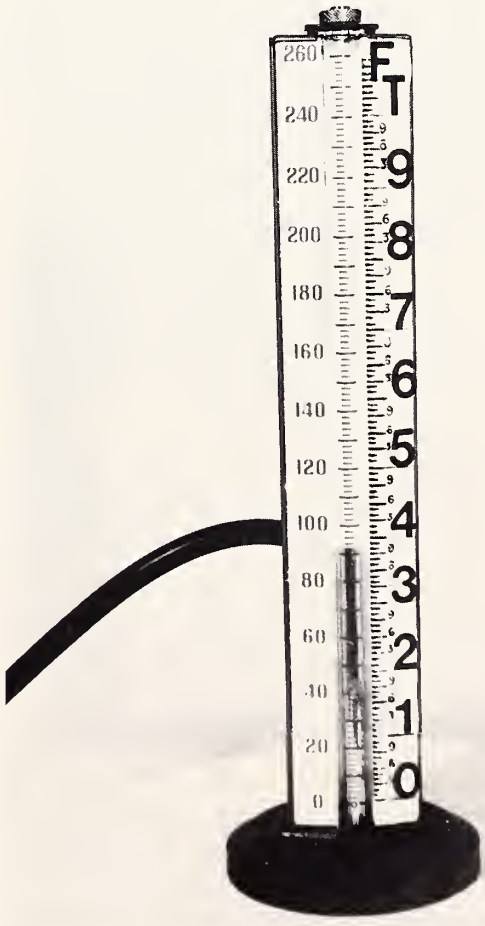


Figure 2. A mercury manometer with an aid to help in the conversion of millimeters of mercury into feet and inches of blood pressure.

feet of blood. If a tape measure were placed up a wall from the floor, the five-foot and three-foot levels may be pointed out as the systolic and diastolic pressures. Youngsters and children appear to understand this method of measuring blood pressure.

Normal bloods vary in specific gravity from 1.048 to 1.066. This suggests a weight or blood pressure/mm variation of 1.8 per cent between these two extremes of specific gravity of normal blood. This method corrects to the normal blood containing its full quota of hemoglobin.

Patients given both blood pressure readings will, perhaps, eventually discard the measurement that is least meaningful to them.

Summary

A method to communicate to the patient his/her blood pressure in both millimeters of mercury and in feet and inches of normal blood has been discussed.

This technique corrects to a normal blood with a specific gravity of 1.066. This may be its best characteristic. Normal human blood specific gravity allows for a 1.8 per cent error between the lightest anemic bloods and the heavier normal bloods.

Acknowledgement

Dr. Douglas Brothers, Chairman of the Department of Physics, and James Ewbank, Chairman of the Department of Mathematics, Benedictine College, contributed valuable assistance in the preparation of this paper.

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Viral Hemorrhagic Fevers

(Continued from page 493)

Special facilities and equipment such as Vickers Isolator for the patient, disposable gowns, special respirator with filter, gloves, and head and shoe covers for hospital staff, and maximum containment laboratory facility (available here at CDC) for handling of clinical specimens are provided by the above governments for any suspected case of these diseases.[19, 20]

As of September 1982, an outbreak of dengue type 1 infection is occurring in Mexico; hence, active virologic surveillance for dengue has been implemented in Texas border towns.[21]

As our understanding of the natural reservoir, pathogenesis, and mode of transmission of many of these diseases is still incomplete, and as there is currently no vaccine or specific treatment for them, they present the medical profession with a challenging enigma. Fortunately a number of investigators both in the United States and abroad are now actively engaged in basic research on these diseases. It is hoped that their efforts will soon bear fruit.

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Toxic Shock Syndrome

A Review

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Toxic shock syndrome, although originally described in children, is more often found in menstruating women. It can be recognized by its clinical course, beginning with a paroxysmal onset of gastrointestinal complaints and hypovolemic shock, followed by other symptoms. Several factors have been suggested that may fit the etiology of the disease, and various recommendations for management have been made.

Toxic shock syndrome (TSS), since its original description, is being diagnosed with increasing frequency. However, since the disease has been recognized only recently, many physicians may be unfamiliar with the disease or have questions about it. Unfortunately, information about this disease is incomplete. This discussion will review the current knowledge of TSS, including definition, a brief history and epidemiology, apparent clinical course, etiology, and current recommendations for management.

Although clinical descriptions of illnesses similar to TSS date as far back as 1927,[1] TSS was first described as a unique entity in November 1978 in a report by J. Todd *et al.*[2] based on seven cases in children aged 8-17 years. Most subsequent cases, however, have been found to occur in menstruating women, as first reported in 1980 by Dean in Minnesota and Davis in Wisconsin. Todd also subsequently reported cases in women. Most of the women were found to be of childbearing age: 80 per cent were under 30 years old.

Between February and May 1980, 43 more cases were reported to the Center for Disease Control (CDC)[3] in persons 13-52 years of age; 95 per cent of the females were in menses at the time of their illness. In all, more than 300 cases were reported in 1980. The Food and Drug Administration (FDA)[4] and others[5] estimate the incidence in the United States to be about three/100,000 women of menstrual age per year. In two states where TSS episodes were closely surveyed, the incidence was reported as 10-15/100,000 women of menstrual age per year in Utah; in Wisconsin, it was estimated at 6.2/100,000.[6] Most cases have been diagnosed in menstruating women, with almost all recurrences occurring in menstruating women. Most of the women with recurrences were using tampons, and *Staphylococcus aureus* was usually isolated from the patient.

Most definitions of the disease are based on descriptions by Todd,[2] and have been modified some-

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what by others, notably Davis *et al.*[6] The latter group used the following as their case definition:

- fever above 38.9C
- documented hypotension or orthostatic dizziness
- palmar or diffuse erythroderma followed by desquamation of the feet and hands
- hyperemia of mucous membranes
- multisystem dysfunction involving at least four of the following: nausea/vomiting; altered state of consciousness; impaired hepatic function; impaired renal function; thrombocytopenia; increased muscle creatinine phosphokinase (CPK); cardiopulmonary dysfunction; or decreased serum calcium and phosphorus
- laboratory studies excluding other infectious etiologies.

Some diseases to rule out include Kawasaki's disease, acute rheumatic fever, scarlet fever, Rocky Mountain spotted fever, leptospirosis, and bacteremia. The definition used by Shands *et al.*[7] differs mainly in that it also requires that cultures of blood, throat, and cerebrospinal fluid samples be negative, but that at least three organ systems be affected instead of four. The CDC uses a similar definition.[4]

The clinical course of TSS is marked by a rather paroxysmal onset of gastrointestinal symptoms (*e.g.*, nausea, vomiting, and diarrhea), fever, hypotension, mucous membrane involvement and rash, all at or near a menstrual period. Volume depletion and hypovolemic type shock may rapidly develop, along with edema. Iannine and Watsky[5] mention that pulmonary edema may be observed with crystalloid fluid replacement after only 24-36 hours of treatment, whereas lesser infiltrates develop with colloid therapy. By days three to four, myocarditis may develop, as determined by cardiac dilatation, EKG changes, an S_3, plus occasional pulmonary vascular congestion. Multi-system involvement is made evident by laboratory findings such as leukocytosis with left shift, elevated BUN and creatinine, sterile pyuria, elevated bilirubin, SGOT, SGPT, elevated CPK, and increased clotting studies. Vaginal cultures are most often positive for *S. aureus*, but interestingly, all other cultures — including blood samples — are usually negative. It has been noted that after five days the patient becomes afebrile and begins to improve, and by ten days after onset, convalescence is completed by desquamation of hands and feet. Recurrence may be noted during subsequent menstrual periods — a less severe, shorter illness. Incidence of such recurrence ranges from 20 per cent[5] to about 28 per cent.[1, 6] In either case, beta-lactamase resistent antibiotic therapy seems to reduce the incidence. Discontinuing the use of tampons also helps; recurrence rates have been reported at 40 per cent when tampon use was continued.[8]

Case fatality rates have been reported to range from 8 per cent[9] to 10-14 per cent.[1, 10, 11] Fatalities in one series[11] were related to recurrent ventricular dysrhythmia and a condition simulating acute respiratory distress syndrome. It appears, however, that the fatality rates will be found to be actually lower as more and more mild cases of TSS are recognized.

Clinical studies have revealed several possible etiologic factors that may be involved with the acquisition of TSS, such as a localized bacterial infection, a toxin, an association with menses, and an association with the use of tampons. The strongest of these associations to date appears to be the presence of a localized *S. aureus* infection in the patients, usually in the vagina. In one series, *S. aureus* was found in vaginal or cervical cultures in 74 per cent of cases,[6] whereas others reported an incidence greater than 95 per cent.[4, 5, 10] Even in those cases not involving menstruating women, a staph infection was usually found or suspected, *e.g.* a wound infection. In contrast, normal vaginal flora have been reported as only 10-15 per cent positive for *S. aureus* in one series,[5] and even less by others.[6, 8, 9] Other culture sites have almost always been negative.

Interestingly, those patients with staph infections all have had no demonstrable bacteremia; thus it has been postulated that a staphylococcal toxin is responsible for the disease. In support of this, certain parallels may be drawn between the manifestations of TSS and symptoms of other toxin induced diseases, such as the erythrogenic toxin in scarlet fever, staph enterotoxin in diarrhea, and diphtheria toxin and myocarditis. Unfortunately, no toxin has yet been isolated that can be consistently related to TSS.

Studies have also revealed a strong association between TSS and menses. Although the disease was originally described in children, a 95 per cent correlation in the female cases between the onset of the illness and the occurrence of menses has been reported.[6] Furthermore, it was noted that recurrences of TSS occurred only in menstruating women, and only during menses.

In relation to this, two groups found significant associations between TSS and the use of tampons. All of the patients in one study[7] had used tampons during the index period of the disease. In addition, the controls were noted to more frequently abstain from using tampons continuously day and night during the menstrual period. Another group[5] reported evidence to suggest that a seasonal occurrence of TSS might correspond to patients using tampons

during physical activities such as swimming. A small study in North Carolina[8] revealed that 71 per cent of patients who had TSS used tampons, as opposed to a usage rate of 26 per cent in controls. However, tampons alone cannot be blamed for the acquisition of illness, because the overall incidence of TSS is low, whereas the use of tampons in United States females is estimated to be 70-80 per cent.[7] A study in New Zealand[12] revealed that although the tampons available there were not sterile, they contained only non-pathogenic organisms; also those tampons would only support the growth of *S. aureus* if soaked in blood.

Management of TSS has been limited to relatively simple steps so far, since concrete risk factors have not yet been identified. Hypovolemic shock is the major concern; thus early treatment should consist of fluid replacement. As previously noted, one group recommended colloid fluids as opposed to crystalloids, to decrease the amount of pulmonary infiltrate that might develop. In addition, the patient should be monitored for complications such as "third spacing" of body fluids, and cardiopulmonary dysfunction. Hospitalization in an intensive care unit may be helpful, and artificial respiratory assistance may be required. Unfortunately, there currently is no specific therapy for TSS, but it is recommended that beta-lactamase resistant antibiotics be administered,[8] even though they apparently have no effect in the treatment of the active disease.[4] However, there is evidence that the use of such anti-staph antibiotics acts to deter recurrence of the illness. It is also recommended that the use of tampons be discontinued until vaginal *S. aureus* colonies have been eradicated. However, no significant differences were noted by one group[7] in the brand of tampon, deodorized tampons, tampons containing highly absorbent materials, frequency of change of tampons, or the amount or dration of menstrual flow.

Summary

Regretably, many questions about toxic shock syndrome remain unanswered, although some answers have been determined. First described in 1978, TSS is thought to have a low incidence in the United States, occurring mainly in menstruating women. It can be recognized by the rapid onset of gastrointestinal complaints, hypovolemic type shock, erythroderma and fever, followed by multisystem involvement. Recovery becomes evident after only about five days in most cases, and convalescence, accompanied by desquamation, requires one to two weeks. TSS has been associated with localized *S. aureus* infection, possible staph toxins, menstruation, and the use of tampons. However, the interrelations, mechanisms, and pathophysiology have yet to be determined. Currently the disease is best managed by copious fluid replacement therapy, antistaph antibiotics, and abstinence from the use of tampons. Still, many questions remain to be answered.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Skin Testing For Penicillin Allergy

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ANTIBIOTICS are used in the treatment of as many as one third of all hospitalized patients. The most frequently administered antibiotics are the beta-lactam antibiotics, including the penicillins and cephalosporins. Eight to fifteen per cent of all hospitalized patients and 3-5 per cent of outpatients claim to have had an adverse reaction to the administration of a beta-lactam antibiotic. The incidence of adverse reactions to penicillin is variously reported as 1-10 per cent. Approximately 2 per cent of all adverse reactions to penicillin are thought to be mediated by immunoglobulin E and are manifested usually by (1) immediate reactions occurring within thirty minutes; (2) accelerated reactions beginning 1-72 hours after administration of the drug; and (3) very rarely, delayed or late reactions beginning 72 hours after drug administration. Ten per cent of these IgE-mediated reactions are life-threatening (anaphylactic) with symptoms such as urticaria, angioedema, wheezing, and shock. Ten per cent of these life-threatening anaphylactic reactions to penicillin are fatal. Penicillin is probably responsible for at least 300 deaths in the United States each year. The incidence of adverse reactions to penicillin appears to be increased when the drug is given via the parenteral route rather than orally. There does not appear to be overwhelming evidence that the incidence is increased in patients with other allergic disease, particularly atopic conditions such as allergic rhinoconjunctivitis, allergic asthma, and atopic dermatitis. There does appear to be an increased incidence of adverse reactions in patients receiving intermittent courses of therapy, especially if the drug is administered in one dose in a depot form.

Immunology

Any of the four types of hypersensitivity reactions originally described by Gell and Coombs may occur following the administration of penicillin. Type 1 reactions are manifested in the form of anaphylaxis, angioedema, urticaria, rhinitis or asthma, and 85 per cent of the time will begin within 15 minutes of administration of the drug. Type 2, or cytotoxic, reactions occur when penicillin is bound to the cell surface of various formed elements of blood inducing the production of IgG or IgM antibodies directed toward the penicillin hapten. Interaction of the antibody with the cell-antigen complex activates the complement system via the classical pathway resulting in cell destruction. Examples of Type 2 penicillin adverse reactions include hemolytic anemia and immune thrombocytopenia. Type 3 reactions occur when antibody to penicillin complexes to penicillin in the fluid phase of blood, depositing in vessel walls or synovial or basement membranes, again activating complement by the classical pathway. Penicillin-induced serum sickness is an example of this type of reaction. Lastly, Type 4, delayed or cell-mediated hypersensitivity, reactions developing 24-48 hours after exposure to the drug may result in the development of various dermatoses.

Penicillin and its many degradation products are small molecular weight compounds (250-400 dal-

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tons) which by themselves are poorly immunogenic (capable of inducing an immune response) and antigenic (capable of binding with an antibody after the immune response has been generated). In order for an immune response to be mounted against penicillin, it must act as a hapten and bind to larger immunogenic proteins. Antibodies of the IgE class are then generated and have specificity for penicillin or its metabolites. These IgE antibodies bind to mast cells and basophils. On future administration of penicillin, the protein-penicillin complex binds to the IgE antibodies attached to mast cells and basophils. This leads to a series of changes in the cell, which result in the release of mediators of immediate hypersensitivity, such as histamine, eosinophilic chemotactic factor of anaphylaxis, neutrophilic chemotactic factor of anaphylaxis, platelet activating factor, and leukotrienes (formerly known as slow reacting substance of anaphylaxis).

When penicillin G (benzylpenicillin) is administered, the major metabolite, accounting for 95 per cent of the drug, is the benzylpenicilloyl moiety. The other 5 per cent of the administered penicillin G either remains unmetabolized or is converted to a number of metabolites such as penicillenic acid, penilloic acid, penicilloic acid, penaldate, penicillamine, penamaldoyl, or penicilloaldehyde. These are collectively referred to as minor metabolites. In actuality, these metabolites are not minor in importance. They are of major importance because antibodies of the IgE class directed to these minor antigenic determinants are responsible for most cases of anaphylaxis. IgE antibodies directed to the major metabolite (benzylpenicilloyl-BPO) are infrequently responsible for anaphylaxis, but more frequently cause urticaria and angioedema. The major antigenic determinant is commercially available from Kremers-Urban Pharmaceutical Company under the trade name Pre-Pen (penicilloyl-polylysine). Unfortunately, the minor antigenic determinants are not commercially available. Preparation of these minor antigenic determinants is too cumbersome for routine clinical practice. However, 90-95 per cent of antibodies directed to these minor antigenic determinants can be detected by skin testing with commercially available penicillin G.

Although the cross-reactivity is frequent between penicillin G and other penicillins or cephalosporins, skin testing with penicillin G and Pre-Pen alone is insufficient for detecting as many as 25 per cent of patients with sensitivities to these other beta-lactam antibiotics. Consequently, when a beta-lactam antibiotic other than penicillin is to be administered, not only should the patient be skin tested with Pre-Pen and penicillin G, but also with the appropriate antibiotic.

History *vs* Skin Test

Simple avoidance of beta-lactam antibiotics in those patients who claim a history of having an adverse reaction to penicillin is inappropriate for many reasons. First of all, two-thirds of all fatal anaphylactic reactions occur in patients who have a negative history for penicillin allergy. Secondly, 70-75 per cent of patients who claim a positive history for penicillin allergy will have negative skin tests to Pre-Pen and penicillin G. If these patients are treated with penicillin, only 0.2 per cent will have an immediate nonfatal reaction to penicillin (2 of 1,019 patients in review of the literature); none of these is life-threatening, and all are easily treated. Thirdly, 6-8 per cent of patients with a negative history for penicillin allergy have positive skin tests. If these patients or the 15-30 per cent of patients with a positive history and positive skin test are treated with penicillin, approximately 45 per cent will develop an immediate nonfatal, or rarely fatal, hypersensitivity reaction.

It is obvious, then, that the history cannot be relied upon in order to determine who should and should not receive penicillin. If it were relied on as the sole guide to predict who would have an adverse reaction to penicillin, then many non-sensitive patients would be denied optimal antibiotic therapy. Conversely, a case could be made for the routine skin testing of all patients about to receive penicillin in order to prevent the 200 fatal anaphylactic reactions per year among patients in the United States who have negative histories for penicillin allergy. However, this poses a significant logistical problem, and we currently recommend skin testing only for those patients about to receive penicillin who have a positive history of a prior adverse reaction to penicillin.

Technique

Skin testing, when done correctly, is a very safe procedure *(Tables I, II)*. The assumption is made that a need for the antibiotic has been definitely established and no other antibiotic will suffice. Informed (not necessarily written) consent must be obtained from the patient, and skin testing must be done in a location where there is immediate access to resusitative equipment and medications. All skin tests should be done on the volar aspect of the forearm so that, in the rare event the patient has adverse systemic symptoms secondary to the skin testing, a tourniquet may be applied to retard absorption of the

TABLE I
SUPPLIES REQUIRED FOR
PENICILLIN SKIN TESTING

25-27 gauge needles
1 ml insulin or tuberculin syringes
Alcohol swabs
Tourniquet
Normal saline for negative control
Histamine 0.1% and 0.01% for positive controls
Penicilloyl-polylysine (Pre-Pen) prepared according to package insert
Penicillin G 10,000 units/ml
Immediate access to resuscitation equipment and medications, including antihistamines and vasopressors

TABLE II
PROCEDURE FOR PENICILLIN SKIN TESTING

- Place all tests in duplicate, on forearm if possible; reproducibility improves reliability of results.
- Do gentle scratch testing with (1) normal saline, (2) appropriate histamine control, (3) Pre-Pen, and (4) penicillin G to rule out exquisite sensitivity.
- If scratch tests are negative, intradermal tests should be applied, using the same reagents.
- If intradermal tests are negative, proceed with administration of penicillin.
- If intradermal tests are positive, choose alternate antibiotic or refer for desensitization.
- Negative histamine controls or positive saline controls invalidate interpretation of other skin test responses. Alternate antibiotic therapy or referral for desensitization should be considered.

skin test antigen into the central systemic circulation. All skin tests must be done in duplicate and the duplicates must agree. If the duplicates do not agree, they must be repeated. Normal saline must be used as a negative control. Histamine should be used as a positive control. For prick or scratch testing, 0.1 per cent histamine phosphate should be used; for intradermal skin testing, 0.01 per cent histamine phosphate should be utilized. Skin test reagents to be utilized should include Pre-Pen (penicilloyl polylysine) at a concentration of 6×10^{-5} M as prepared via the instructions accompanying the reagent, as well as sodium penicillin G at a concentration of 10,000 units/ml. The patient should be skin tested initially by the scratch method. This leads to the introduction of smaller volumes of reagent than does the intradermal method, and is therefore safer for the patient who may have exquisite sensitivity. A 26-gauge needle is used to make several short superficial scratches that barely break the skin and never draw blood. A drop of skin test material is then placed over the scratched area, and in one or two minutes is blotted gently away. There should be six such scratch tests done — one positive control, one negative control, two Pre-Pen tests, and two penicillin G tests. If a wheal (induration) greater than 3 mm in diameter develops and is still present at 15-20 minutes, the test is considered positive. If both tests for Pre-Pen and both tests for penicillin G are negative, intradermal skin testing is undertaken. There should be five such intradermal skin tests — a positive control with the 0.01 per cent histamine, two Pre-Pens, and two penicillin Gs. Skin tests are done in a fashion very similar to mantoux TB skin tests using a tuberculin syringe. However, the usual volume of 0.1 ml that is administered with standard tuberculin skin testing is not employed; rather a volume of 0.02-0.04 ml is injected sufficient to create a bleb of 3-4 mm diameter in the epidermis. A positive skin test is considered to be present if there is 5 mm or greater wheal diameter present at 15-20 minutes. With both scratch and intradermal skin testing, erythema does not influence the decision as to the positivity or negativity of the skin test. If a beta-lactam antibiotic other than penicillin G is to be administered, skin testing with the appropriate antibiotic should be undertaken, initially by the scratch method, at a concentration of 0.25 mg/ml, then 2.5 mg/ml. If negative, then the intradermal method should be utilized with the same concentrations.

Interpretation

If the skin tests are negative, the patient may be given the appropriate antibiotic with less than 1 per cent chance of even mild immediate hypersensitivity reactions. If the skin test is positive the antibiotic should be avoided if at all possible. If administered, there is a 45 per cent incidence of an immediate hypersensitivity reaction. If it is still thought necessary that the implicated beta-lactam antibiotic be administered, an infectious diseases consultation should be obtained to review the need for the antibiotic and to assist with desensitization if therapy with this antibiotic is mandated. These skin tests are in no way helpful in predicting the susceptibility to the development of other adverse, non-IgE-mediated reactions such as hemolytic anemia, immune thrombocytopenia, interstitial nephritis, serum sickness, drug fevers, or various maculopapular skin eruptions. Such penicillin skin testing is also not useful in evaluating in a post hoc fashion whether a suspected drug reaction is due to penicillin or not. This is because both false-positive and false-negative skin tests may occur. False-positive skin tests may occur

because it is known that some patients will develop IgE antibodies to penicillin during a course of penicillin therapy; these antibodies do not cause an adverse reaction because the patient concomitantly develops IgG blocking antibodies. Conversely, a false-negative reaction can be seen in someone who has had an IgE-mediated reaction to penicillin; mast cells and basophils have been depleted of mediators of immediate hypersensitivity as a result of the immediately preceding reaction, leading to a false-negative skin test. Importantly, the act of skin testing itself has not been reported to induce penicillin sensitivity. It is clear that the penicillin allergic state is not static, but rather waxing and waning with time. Consequently, the results of skin tests done at one point in time cannot be used to predict the patient's likelihood of sensitivity at a later date. A patient with a known history of penicillin sensitivity or a known history of positive penicillin skin tests should be retested upon each subsequent exposure to the antibiotic.

Summary

The immediate hypersensitivity reactions mediated by IgE are a relatively common problem upon administration of beta-lactam antiobiotics, such as penicillin. One cannot depend upon the history of adverse reactions to penicillin to predict the sensitivity of the patient. This is reliably predicted and safely, as well as easily, performed with skin testing initially by the scratch and then the intradermal (if necessary) routes. All patients with a positive history of adverse reactions to penicillin should have such skin testing prior to the administration of each course of penicillin therapy.

Self-Assessment Questions

1. A prior history of adverse reactions to penicillin is a good predictor of allergic reactions with subsequent use of the drug. (T or F)
2. Intact penicillin molecules are responsible for most penicillin reactions. (T or F)
3. A positive scratch test to penicilloyl-polylysine or to dilute penicillin G suggests exquisite penicillin reactivity. (T or F)
4. Penicillin skin tests predict such reactions as drug fever, serum sickness, hemolytic anemia, granulocytopenia, interstitial nephritis, and maculopapular rashes. (T or F)
5. Corticosteroids or antihistamines will not suppress positive skin test results. (T or F)

(Answers on page 510)

DRUGS, ALCOHOL, AND YOUNG ADULTS

All three Alcohol, Drug Abuse, and Mental Health Administration Institutes, and the Office of the Administrator, have begun working on an urgent problem: namely the growing U.S. population of young adult chronic mental patients who are abusing alcohol and drugs.

A cross-agency planning committee has met to lay the groundwork for an ADAMHA initiative on this issue. The first step will be a working conference of invited experts to be held shortly. The conferees will pinpoint the unique characteristics and treatment needs of this young population. On the basis of their conclusions, the agency will report to the field on ways to improve care of such patients.

In a brief paper outlining the scope of the problem, the NIMH Division of Mental Health Service Programs stated: "It is the combination of medical-psychiatric-substance abuse and behavioral problems that significantly distinguishes these patients from most other populations of both chronic and acute patients.

"Many are socially isolated and have limited or poor support systems. . . . Some have brain damage or suffer from flashbacks. Generally, they are quite vulnerable to stress; they often have poor judgment and do not learn from experience. . . .

"A number of studies indicate that well over half were in contact with some kind of psychiatric treatment facility before the age of 18 years, and about half have had multiple hospitalizations."

Because young adult chronic patients need a broad range of specialized and support services, the agency will be looking for ways to involve many different types of service providers, including emergency services, mental health clinics, alcoholism and drug abuse treatment services, public welfare agencies, and law enforcement agencies.

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Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Starch Blockers

Question: Will you provide information on the use and current status of starch blockers?

Answer: Starch blockers have been available as non-prescription products under such names as Starch Block, Les-Starch, Blok-Cal, Alpha-Plex, Seirolac, and Sta-Trim to name a few. These products were promoted as a "real breakthrough" in diet therapy and thus became very popular with the dieting public. This popularity resulted from claims of weight loss with the use of these products, without decreasing starch intake.

The active ingredient in starch blockers, referred to as amylex, NBE (Northern Bean Extract), or starch blocker, is a processed derivative of beans, such as kidney and northern beans.[1, 2] Amylex presumably forms an insoluble complex with the pancreatic enzyme alpha-amylase. Alpha-amylase is responsible for the breakdown of starch to maltose which is eventually converted to glucose. The amylex/alpha-amylase complex passes from the gastrointestinal tract along with ingested starches, thus preventing the absorption of starch calories. These products make no claim of decreasing the absorption of calories resulting from fat, protein, or sugar.

Robertson, in a recent article on starch blockers, raises three questions that exemplify the concerns of the medical community regarding the use of these products as diet aids:[3]

1. Is there any authentic published data on a large number of patients to substantiate the claim that five of seven patients are successfully losing weight while using this product?
2. Is there any information on long-term side effects of blocking the enzyme amylase?
3. If this new compound-amylex is discharged in the feces, what else gets pulled out with this insoluble complex such as sodium, potassium, other minerals, and trace elements?

Unfortunately there are no published, controlled studies to substantiate claims of weight loss when taking starch blockers. Also, studies addressing long-term effects or loss of vitamins, minerals, and other nutrients are not available.

The concept of slowing, or decreasing, carbohydrate absorption with the use of enzyme inhibitors has been studied by few investigators. Jenkins and associates studied the absorption of carbohydrates following the administration of acarbose, an alpha-glucosidase inhibitor, in 13 healthy male volunteers.[4] They found 200 mg of acarbose reduced the area under the blood glucose curve by 89 per cent following the administration of sucrose and by 80 per cent following the administration of starch. The area under the blood glucose curve did not change significantly following the administration of glucose or maltose with acarbose. Gerard and associates studied the use of acarbose (100mg three times/day) in 28 insulin dependent diabetic patients for a two month period.[5] During the study, the daily caloric and carbohydrate intake of patients remained constant. They found that neither weight nor insulin requirement changed significantly during the study. However post-breakfast and post-dinner blood glu-

(Continued on page 509)

The President's Message

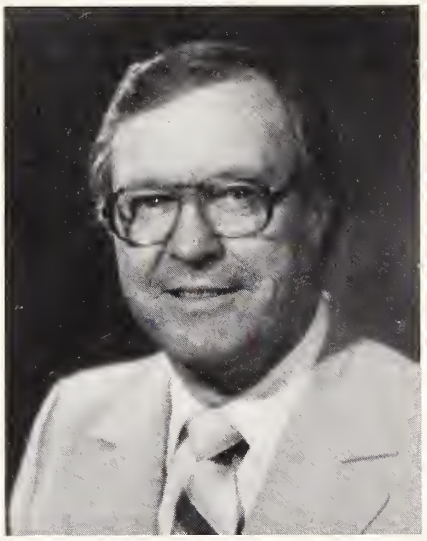
Substance abuse in Kansas and in the United States has become a significant social and medical concern. Increasingly it seems to be a contributing factor in many of the problems we as physicians deal with in our office and hospital practice.

The influence of alcohol, illegal drugs, or abuse of prescription drugs is evident in most accidents, incidents of child and wife abuse, and most other social and family disruptions.

The total cost of alcohol abuse in the United States last year was estimated to be $60 billion. In contrast, the nation spent only $64 billion on heart disease and stroke.

The incidence of substance abuse at the junior high as well as the senior high school level is alarming. The tragic aspect of this is that excessive alcohol ingestion or the use of marijuana, cocaine, and other such illegal drugs are much more detrimental to the developing nervous system than they are to the mature system. The impairment of some of the higher intellectual functions in humans, such as social behavior, interpersonal relationship, motivation, and planning for the future is evident in long time users, especially the young. The 15- to 24-year age group is the only age group with a rising death rate today, and many of these deaths are drug related.

The implication of this type of behavior by a substantial segment of our population is tremendous,



especially when viewed in terms of health and social costs, as well as national defense.

As physicians, we need to become interested in this problem and learn as much about it as possible. We need to have input into our communities as they attempt to deal with this issue.

The Kansas Medical Society, with our Auxiliary and other concerned and interested organizations, are in the process of planning a drug conference for April 8-9, 1983, in Salina. We hope that you will have the time and interest to attend.

Sincerely,

President



A Matter of Opinion

It is unreliably reported that the pictures on the walls of a remote cave in Somali tell of the beginnings. It seems that Oog and Mugh, once they were sated with food, sleep and mating, completed their transition to the fully human state by deciding to organize the Caveman's Benevolent and Protective Society. At once different attitudes emerged with some of the objectors choosing to join up but continue their obstreperations within, while others sought other caves where they could exercise their isolated independence. The impulse to organize was not bred out of the latter, however. Some of them formed their own organization which was anatomically identical to the CMBPS but functioned by consistent reaction against it. There were, eventually, a few who were unable to tolerate even this degree of union and retired to still more remote caves where they survived precariously, their only accomplishment being the propagation of like-minded cavemen whose progeny survive even to this day.

Undaunted, Oog and Mugh went about their business of setting up membership requirements and assessing dues and, as the first order of business, appointed a committee (over some objections, it is true) to find out how they were doing and, in particular, why anyone would not choose to be a part of their project.

We can report reliably that their spirit is alive and exists in forms they never dreamed of. The one that interests us at the moment (and most of the time) is the Kansas Medical Society. Thus it was that, in what might be considered a gesture of CMBPS recertification, the KMS commissioned a membership survey. This was done with proper obeisance to the spirits of O. and M.: conducted by properly initiated celebrants of the mysteries of opinion-taking, telephonic scrutiny of the subjects, and appropriate electronic distillation of the testimony. The purpose was laudable and the results sufficiently informative to justify the effort and still not bring down the walls of the cave. In the best CMBPS traditions, the exercise sought to find out how we were doing and where we should go.

This should, of course, be known to 98 per cent of KMS members since that portion admitted to reading the KMS *Newsletter,* which reported the results in August. On the other hand, only 80 per cent acknowledged finding the *Newsletter* useful, so apparently the effort was lost on some 12 per cent. At the same time, only 77 per cent reported reading the *Journal,* so these comments will be lost to some 21 per cent. We did pick up a few more since 80 per cent confessed to "browsing," but we doubt that the effort to get through one of these editorials can be classified as browsing, so we'll write off that 3 per cent.

Not to derogate the purpose, methods, or findings of the study, the survey approach does offer a certain perspective: it is often more interesting for what it does not say than for what it does. Moreover, while we're pleased to have documentation for the positive feelings of the membership for the Society's overall efforts (and our own in particular), we don't want to be too satisfied. After all, if the results had indicated that no one loved us, we'd be working up all sorts of reasons to discredit it, so objectivity does call for a little leavening. Without discounting the agreeable features of the study then (and with passing thanks to the 77%), we have been reflecting on the hidden meanings in the whole thing.

As was reported, an aliquot of 400 members of the KMS was selected for the study. Since the response rate was reported to be greater than 50 per cent, this translates into the fact that the study was based on direct contact with something more than 200 individuals (not to overlook the complementary fact that

almost as many declined the honor). Inasmuch as the 1981 membership of the KMS was 2800, our statistical expertise tells us that the results were derived from about 7 per cent of the membership. The opinion analysis industry has come a long way since the *Literary Digest* debacle, so we accept the premise that current methods can indeed produce valid results from such a small sampling. Still, we can only hope the 93 per cent not questioned are of the same (proportionate) mind.

Implications of skepticism aside, our interest is of a different order. Physicians licensed by the Kansas State Board of Healing Arts totaled 6329 in 1981. The Board reports that only 2957 were actually practicing in Kansas. This means that only 157 eligible physicians in the state were not members of KMS. Frankly, this information set us back a bit as we were about to comment on the number of physicians practicing in Kansas without the blessings of KMS membership. We had assumed that the state situation paralleled that of the AMA and, as is well known, less than one-half of the nation's total physician population belongs to the AMA (a point it finds annoying but which, to the best of our knowledge, it has been able to use to advantage only once: when accused of constituting a medical monopoly, it responded, rather futilely as it happened, that this could hardly be the case with fewer than one-half of the physicians belonging). We were prepared to speculate on why so many Kansas physicians do not belong to the KMS only to discover that we were talking about a little more than 5 per cent, which we could hardly turn into stimulating information, even by our relaxed statistical methods. Lesser souls might have considered the show over since there didn't seem to be much to talk about, but that is what sets editorial writers apart from the common people: that is where *they* start. We'll note, rather, that the study takes on a brighter polish since it provides (if we accept it at all) a very creditable assessment of organized medicine in Kansas in the opinion of its own members.

There is still a question, however, of what happens at the higher levels of organization. We mentioned that fewer than one-half of the eligible physicians in the country are members of the AMA. Kansas physicians, however, seem to find the AMA climate congenial since some 70 per cent of KMS members are also AMA members. We assume that the 30 per cent chose, for whatever reasons, not to participate at the national level. It is the "whatever reasons" at whichever level that should be of as much interest to the organizations as the features that get good marks on the surveys.

It seems unlikely that failure to join organized medicine is simply a passive matter — a case of never-got-around-to-it. Given the publicity attending its activities and the visibility of medical functions on its various organizational levels, no physician can help but be aware that much is being done in the name of medical practice, although some manage to find objectionable features in all. In seeking to avoid any identification with the medical establishment, the dissenters become increasingly strident in proclaiming their independence. But this is not to categorize all who reject the general organization life as mindless malcontents. Theirs can be a valid and effective approach depending upon the integrity of the objector. One wonders, however, how they can expect their fragmented efforts to have constructive effect on the many matters of medical service that must be mediated through combined efforts of medical expertise. It is certain that not every action and posture of the AMA — or even KMS, as is now documented — is totally agreeable to all members, but such is the nature of the democratic process, still the most effective use of masses of human intellect and energy yet devised. The fact remains, however, that a certain number of physicians simply do not choose to be a part of the process.

Beside this dissenting attitude, however, there is an area of medical practice that has, perhaps unintentionally, contributed to decreased attention to the general medical organizations — specialization. From its pre-Civil War beginnings, the KMS sought to enlist all legitimate physicians, if for no other reason than the fact that there weren't too many anyway. The annual meetings presented a variety of subjects and included social events of high order demonstrating the pleasure of widely dispersed physicians in getting together with their own kind, if only once a year. This, in a sense, was a repeat of the process that originated in the first areas of settlement at an earlier time, and moved westward as the country opened up. As specialization developed, the general organizations sought to keep pace by having specialty "sections" to accommodate. But increasingly, specialties — and now subspecialties — have focused on their own interests with growing tension on the umbilical cord. Many specialists feel their interests are better served by their own societies and reserve their limited time and energies for them. This may be less true at state level — or more so — we don't know. But it is true that even if some of these individuals accept membership in the state or national societies, their attention to them is often marginal, being given rather to their specialty societies.

Parenthetically we can note that this is reflected in the journals produced by the state societies. We acknowledge our good fortune in having a sufficient number of contributors to keep us supplied with manuscripts, but a significant number of specialists prefer the prestigious level of their specialty journals for publication. This is a major reason why many state periodicals have ceased to publish or have turned to socioeconomic papers and general medical news.

Another segment of medical personnel that has been insufficiently represented at the organizational level is women physicians. Whatever the deficiency in numbers, the greater loss has been in the effect. How much of their absence has been due to neglect by their male colleagues, how much due to frank prejudice (both ways), and how much due to personal accommodations to domestic and social pressures is impossible to say, but all are diminishing. Concomitant with the real and sustained interest of organizations to enlist women members is the obvious increase in numbers of women physicians available. It is a mutual response: as the organizations experience the benefits of their increasing presence, women physicians are recognizing that they can achieve equitable recognition and reward for service.

The fact that the proportion of Kansas physicians not participating in the state society is so small means that this fact can offer little in the way of direction the organization might take to enroll them — or whether it is perhaps better to leave them there. More importantly, it suggests that the philosophy of the Society is compatible with that peculiar ethnic, urban-rural, industrial-agricultural, labor-professional mix that is Kansas. It is unlikely that the stability of the state will ever be matched on a national level or that the AMA could ever achieve a comparable level of service or approval. Still, we can recommend the survey system to them — it turned out better than we expected. — *D.E.G.*

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Starch Blockers

(Continued from page 504)

cose values decreased by 25 and 24 per cent respectively. Daily mean blood glucose levels also decreased by 18 per cent. The most common side effect reported in both studies was flatulence.

Starch blockers were marketed in the United States as special dietary *foods,* thus not subject to Food and Drug Administration (FDA) controls governing the marketing of drugs. Recently (July 1982) the FDA asked manufacturers and distributors of starch blockers to voluntarily discontinue marketing such products until scientific data confirming safety and efficacy is submitted.[1] This action resulted from complaints of nausea, vomiting, diarrhea, and stomach pains received by the FDA, as well as concerns of long-term effects of starch blockers.

In response to the FDA's request, several manufacturers and distributors have filed suits claiming their products are labeled and distributed as foods in compliance with all applicable federal laws and regulations.[6] Until the controversy is resolved, the public will continue to seek information from physicians and other health professionals as to the safety and effectiveness of starch blockers. At this time there is not enough scientific data to substantiate the safety and effectiveness of starch blockers.

Submitted by
Bruce Scott, R.Ph.
Pharmacy Resident

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

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Pulmonary Amyloid Complications

(Continued from page 487)

2:1.[4] Cough, dyspnea, and hemoptysis are the chief complaints of patients with tracheobronchial deposits, and are manifested by wheezing and rhonchi on examination. Stridor and hoarseness have been mentioned in about 33 per cent of cases.[11] Abnormalities on chest roentgenograms are frequent and include atelectatic changes distal to the site of obstruction in 56 per cent,[12, 13] while hilar and mediastinal masses are less frequent.[14] Abnormal bronchographic patterns have also been mentioned,[15] and pulmonary function studies usually reflect the severity of air flow obstruction and degree of hyperinflation.

The first reported cases of tracheobronchial amyloidosis were diagnosed at autopsy or from resected specimens at thoracotomy. At present, the diagnosis is best established by bronchoscopy which has been successful in most reported cases.[11] Although transbronchoscopic resection of obstructing amyloid lesions can produce temporary or even long lasting relief,[16] the risk of fatal hemorrhage[17] is rather high and careful consideration should be given to this fact when such a diagnostic or therapeutic procedure is contemplated. Other varieties of pulmonary involvement in systemic amyloidosis include pulmonary nodules,[18, 19] single or multiple,[20] which are usually discovered as an incidental roentgenographic finding, and subsequent surgical resection (for suspected malignancy) has resulted in cure.

A transthoracic needle biopsy[21] may be a useful procedure in selected cases, and obviates the necessity for thoracotomy to diagnose the nodule or diffuse version of pulmonary amyloidosis. The diffuse parenchymal pulmonary amyloidosis[22] carries the worst prognosis and usually leads to death from respiratory insufficiency due to alveolar septal infiltration[23] and impaired gas transfer.

Spontaneous regression of diffuse tracheobronchial amyloidosis is exceedingly rare and has been described in only one recent case report.[24] Despite extensive laboratory investigations, the cause of tracheobronchial amyloidosis is unknown. Amyloid fibrils from patients with primary amyloidosis consist of fragments of immunoglobulin light chain called amyloid light chain (A-L) protein. This has been confirmed in vitro by Glenner[25] *et al.*, who produced characteristic amyloid with digestion and thermal treatment of whole and fragmented light chains. Biochemical studies on amyloid fibrils isolated from patients with tracheobronchial amyloidosis had the N-terminal amino acid residue identical to the variable region of immunoglobulin light chains[26] similar to that found in primary amyloidosis.[27] Although it has been suggested that tracheobronchial amyloidosis may be due to increased presence or production of light chains in bronchial secretions and increased degradation or release of excess light chains by alveolar macrophages, the exact mechanisms are not clearly identified and suggest the need for further prospective studies.

Conclusion

Generalized amyloidosis frequently involves the lower respiratory tract and may lead to impairment of pulmonary physiologic functions. Fiberoptic bronchoscopy and needle lung biopsy are valuable tools for establishing the diagnosis in this rare entity.

References are available from the authors.

Penicillin Allergy

(Continued from page 503)

Answers

1. *False*. Seventy-five per cent of patients with prior penicillin reactivity will have negative immediate skin tests, and can, therefore, receive the drug safely.
2. *False*. The penicilloyl determinant causes most allergic reactions. Penicilloyl-polylysine (PrePen) tests for reactivity to the major determinant in penicillin allergy, while use of dilute penicillin G screens for reactivity to minor determinants.
3. *True*.
4. *False*. Skin testing predicts IgE mediated reactions (immediate hypersensitivity) such as anaphylaxis, angioedema, asthma, or urticaria.
5. *False*. Although moderate doses of corticosteroids (less than 60 mg prednisone daily) will not suppress skin test responses, antihistamines will blunt the wheal and flare reaction.

Suggested Readings

1. Adkinson, Jr., N. F.: A guide to skin testing for penicillin allergy. *Res. Staff Physician* 23:55-59, 1977.

2. Adkinson, Jr., N. F.; Thompson, W. L.; Maddrey, W. C. and Lichtenstein, L. M.: Routine use of penicillin skin testing on an inpatient service. *N. Engl. J. Med* 285:22, 1971.

3. Green, G. R.; Rosenblum, A. H. and Sweet, L. C.: Evaluation of penicillin hypersensitivity: Value of clinical history and skin testing with penicilloyl-polylysine and penicillin G. *J. Allergy Clin. Immunol.* 60:339, 1977.

4. Solley, G. O.; Gleich, G. J. and Van Dellen, R. G.: Penicillin allergy: Clinical experience with a battery of skin-test reagents. *J. Allergy Clin. Immunol.* 69:238, 1982.

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Estrogen/Postmenopausal Women

NIH Consensus Development

A Consensus Development Conference on Estrogen Use and Postmenopausal Women was held at the National Institutes of Health in 1979. Risks and benefits of estrogen use, which are of great interest to the nation's 31 million postmenopausal women and their physicians, were explored at the meeting.

Among the subjects addressed were the benefits of estrogen use, including treatment for menopausal symptoms and possible prevention of osteoporosis; hazards of estrogen use; relative risks and benefits of various types of estrogen therapy; economic issues; and indications for and contraindications to estrogen use.

At NIH, consensus development conferences bring together biomedical research scientists, practicing physicians, consumers, and others as appropriate, in an effort to reach general agreement on the safety and efficacy of a medical technology. That technology may be a drug, device, or medical or surgical procedure.

Three position papers were prepared for the conference. These, plus the response of the panel, general discussion by the audience, and an analysis by the panel and other invited conference participants contributed to the conclusions presented below.

In approaching the topic, the group first reviewed evidence for the efficacy of estrogens in treating specific conditions associated with the menopause. It was accepted that estrogens are more effective than placebo in decreasing the frequency and severity of vasomotor symptoms (hot flashes and sweating). Questions that remain to be answered include whether vasomotor symptoms represent a homogeneous entity with a single cause and why some patients require much larger doses than average to control symptoms. There was general agreement that the decision of whether to initiate therapy should depend on the severity of symptoms and the patient's perceived need for relief, and that the lowest effective dose should be utilized. The occurrence of hot flashes naturally declines over a period of time, and unnecessary prolongation of therapy should be avoided.

It is recognized that estrogens are effective in overcoming the atrophy of the vaginal epithelium and associated symptoms, which may include dryness, burning, itching, and pain during intercourse. It was suggested that the possible relationship of urinary tract symptoms to estrogen lack be more thoroughly investigated. Attempts to avoid systemic effects by treating vaginal symptoms with local application of estrogen-containing creams have been common. However, evidence now exists that the estrogens in these creams may be absorbed rapidly into the bloodstream. The biological consequences of this absorption are undetermined and require study.

There is no evidence at present to justify the use of estrogens in treatment of primary psychological problems. Surveys have shown no established specific or temporal association of sleep patterns, mental performance, mood, or psychological state with menopause or estrogen deprivation. On the other hand, in preliminary intervention studies comparing estrogens to placebo, effects on sleep latency and REM sleep have been noted. Some improvement in mental well-being in women receiving estrogens may be secondary to alleviation of physical symptoms.

The group acknowledged the validity of three ramdomized trials indicating that exogenous estrogens can retard bone loss if given around the time of the menopause. Except for dietary calcium, which appears to decrease bone loss to a lesser extent, other substances have not been shown to have such an effect. It is inferred but not proven that this retardation of bone loss will prevent the ultimate development of osteoporosis and attendant fractures. Case-control studies not yet published but discussed at the meeting report an association of estrogen use with a decreased risk of osteoporosis-related fractures. However, more data are needed before the efficacy of estrogens in preventing fractures can be established. An inconsistency was noted: namely, in the randomized trials, accelerated bone loss following discontinuation of estrogen use resulted in loss of any favorable effect on bone mass, whereas in the case-control studies, use of estrogens at any time in the past conferred some protection against fractures. Identification of patients at increased risk of osteoporosis would be desirable because of the strong possibility of successful prophylaxis. One high-risk group in which to investigate possible benefits of estrogens consists of patients who already have developed osteoporosis and sustained fractures. Estrogen administration represents a promising approach to prevention of the wide-

spread problem of hip fracture.

There is no convincing evidence that estrogens in customary doses increase the risk of thromboembolic phenomena, stroke, or heart disease in women who have undergone natural menopause. Although it was once hoped that estrogens would protect against heart disease in aging women, this effect has not yet been demonstrated. One promising approach would be to devise a more physiological mechanism for estrogen replacement. Because oral therapy results in the delivery of supraphysiological concentrations of estrogens to the liver, it can exert an exaggerated effect on lipoprotein metabolism, blood coagulation, and other important processes.

The group reviewed the evidence for adverse effects associated with postmenopausal estrogen use. In the absence of exogenous estrogens, the incidence rate of endometrial cancer is approximately 1/1,000 postmenopausal women per year. It was recognized that this rate increases several fold beginning after approximately two to four years of use of 0.625 or 1.25 mg of conjugated estrogens per day. Evidence was presented that the risk of endometrial cancer increases with the duration of use and declines after discontinuation. Estrogen use is most strongly associated with lesions of the lowest grade and earliest stage. Of interest is the temporal relationship of the number of estrogen prescriptions and the incidence of carcinoma of the endometrium: both rose steadily until 1976 and then declined in parallel. Although the incidence of carcinoma of the endometrium rose, mortality from the disease did not increase. A considerable part of this discrepancy may be attributable to early detection and the high cure rate.

Cystic hyperplasia of the endometrium, which is considered a premalignant condition, has been associated with unopposed estrogen, whether endogenous (as in anovulatory states) or exogenous. The cost effectiveness of sampling the endometrium in order to screen for endometrial hyperplasia and cancer in completely asymptomatic women currently or potentially receiving estrogens is uncertain at present. It was agreed that suction curettage is effective in evaluating the endometrim and that the cause of any bleeding must be determined. A report indicating that uterine bleeding may sometimes be absent early in the course of endometrial cancer was presented at the meeting. Hence, prudence would suggest that, even in the absence of bleeding, the endometrium should be sampled before and during estrogen therapy on a yearly basis.

The use of progestins for several days of each estrogen treatment cycle has been demonstrated to decrease the occurrence of endometrial hyperplasia and might also reduce the associated risk of developing cancer of the endometrium. Before the application of combined therapy becomes established, risks of the various progestins must be adequately evaluated.

The association of estrogens and breast cancer in experimental animals is well known. Careful review of several well-conducted case-control studies has not revealed such a relationship in humans. In two followup studies of estrogen users, varying associations were encountered. Compared to data on the general population, one showed an excess of cases in years five to nine, but the other only after 15 years, of estrogen use. Incidence rates of breast cancer have not changed in parallel with those of estrogen use, as have those of endometrial carcinoma. Because of the high incidence and relatively poor prognosis of breast cancer, any possible association with estrogen use remains a concern.

There are experimental data indicating that estrogens can induce the production of lithogenic bile, and in one study a 2.5-fold relative risk for the development of surgically confirmed gallbladder disease was observed.

One area of general agreement was that the patient should be given as much information as possible about the evidence for the effectiveness of estrogens in treating specific menopausal conditions and the risks that their use may entail. Patients must be kept continually informed of new findings as they arise. Given the current state of knowledge, no general recommendation applicable to all postmenopausal women can be made.

Some concern was expressed about women who have undergone menopause many years in advance of the normally expected age. Although most of the participants felt intuitively that approximation of the normal physiological state through hormone replacement therapy would be best, there are no carefully controlled studies comparing the risks and benefits in these circumstances. Support was voiced for conserving the ovaries of young women when possible.

General consensus was reached on the current state of knowledge, as described. It is clear, however, that much additional information is needed. Specifically, we need systematic knowledge of the natural course of the menopause in the absence of hormonal therapy, of alternatives to estrogen use, of the optimal way to provide estrogens if they are to be used, and of all aspects of their beneficial and adverse effects. Special attention should be directed

(Continued on page 513)

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References: **1.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **3.** Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther 21*:355-361, Mar 1977. **4.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **5.** Meyer JA, Kurland KZ: *Milit Med 138*:471-474, Aug 1973. **6.** Feffer HL, Gibbons B: *Med Times 101*(8):130-135, Aug 1973. **7.** Jacobson A *et al*: *Psychophysiology 7*:345, Sep 1970. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Kales A, Scharf MB, Kales JD: *Science 201*:1039-1041, Sep 1978. **10.** Kales A *et al*: *JAMA 241*:1692-1695, Apr 1979. **11.** Monti JM: *Methods Find Exp Clin Pharmacol 3*(5):303-326, 1981.

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Estrogen/Postmenopausal Women

(Continued from page 512)

toward studies that can determine the proper use of estrogens in young women having undergone oophrectomy ten or more years before the natural time of menopause. No general formulation regarding therapy can be given. Rather, each individual patient must base her decision on the relative values that she assigns to relief of symptoms, to expectations for optimizing health and well-being, and to various risks sustained in the process. Socially and culturally based attitudes toward the menopause may influence these values and require further definition.

The Consensus Development Conference on Estrogen Use and Postmenopausal Women was sponsored by the National Institute on Aging, assisted by the Office for Medical Applications of Research, NIH.

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Practice in Living

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ANUSOL-HC® Suppositories/ ANUSOL-HC® Cream

Before prescribing, please see full prescribing information. A Brief Summary follows.

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in external and internal hemorrhoids, proctitis, papillitis, cryptitis, and fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

CONTRAINDICATIONS

Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

WARNINGS

The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

PRECAUTIONS

General

Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

DOSAGE AND ADMINISTRATION

Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

Store between 59°-86°F (15°-30°C)

1089G010

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(Continued from page 475)

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See next page for brief summary of prescribing information.

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Dental pain and episiotomy pain are predictable, reproducible "standards" that make possible objective comparisons of effectiveness of different analgesic agents.

- Measured against 15, 30 and 60 mg doses of codeine phosphate in a double-blind study of 287 patients, 400-mg doses of ibuprofen proved "significantly better than codeine on almost all pain intensity, degree of relief and duration of analgesia parameters."[2]
- Measured against a propoxyphene-acetaminophen combination for pain relief after 3rd molar extractions, ibuprofen proved equally effective and caused fewer side effects. Ibuprofen was associated with faster recovery, evidenced by more rapid reduction of trismus and return to normal function.[3]
- Measured against post-episiotomy pain in 30 patients, "ibuprofen was effective in treating the swelling as well as pain...during the first and worst days. Therefore, it is not only the analgesic but also the anti-inflammatory effect of ibuprofen that are the beneficial factors..."[4]

Measure RUFEN® (ibuprofen) against any mild to moderate pain

RUFEN	Acetaminophen + codeine combinations
• single-entity, peripheral-acting analgesia	• combined drugs act partly through central opioid pathways
• powerful treatment of both pain and inflammation	• virtually no treatment of the inflammatory component
• better tolerated than aspirin	• combined side effects of two drugs—warning required about driving or operating machinery; possible respiratory depression with alcohol, tranquilizers, other common medications
• no narcotic risk, red tape, records	• narcotic precautions required
• matchless economy in a modern NSAID	



References:

1. Hart FD, Huskisson EC, Ansell BM in Hart FD (editor): Drug Treatment of the Rheumatic Diseases, 2nd Ed, Adis Press, Balgowlah, Australia, 1982, p. 30.
2. Rondeau PL, Yeung E, Nelson P: Canad Dent Assoc J 46:433-439, 1980.
3. Selwyn P and Giles AD: Br Jrl of Clin Practice, Supplement 6, Safe and effective analgesia following dental surgery: A comparison of brufen and distalgesic. Pg 87-90. 1980.
4. Taina E: Curr Med Res Opinion, 7:423-428, 1981.

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And Rufen® Measures Up Best

RUFEN® (ibuprofen) Tablets

INDICATIONS AND USAGE: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in the long-term management of these diseases. Safety and effectiveness have not been established for Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain. Treatment of primary dysmenorrhea.

CONTRAINDICATIONS: Patients hypersensitive to ibuprofen, or with the syndrome of nasal polyps, angio-edema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory drugs (see WARNINGS).

WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination.

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen.

DRUG INTERACTION: *Coumarin-type anticoagulants.* The physician should be cautious when administering Rufen to patients on anticoagulants.

Aspirin. Concomitant use may decrease Rufen blood levels.

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers.

ADVERSE REACTIONS: Incidence greater than 1%. Gastrointestinal: The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS). *Incidence 3% to 9%.

Incidence less than 1 in 100. Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses:* hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) [see PRECAUTIONS]. Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown. Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

OVERDOSAGE: Acute overdosage, the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d.

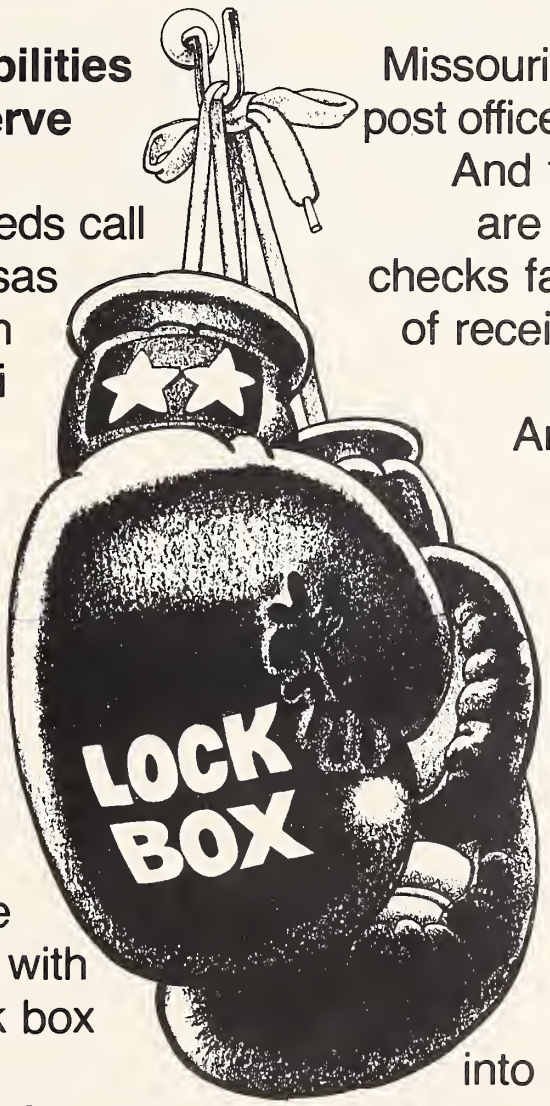
Dysmenorrhea: 400 mg every 4 hours as necessary.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription.

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Continuing Medical Education

The 1976 Kansas Legislature enacted a law requiring continuing medical education as a requisite to relicensure in Kansas. HB-2723 provides that, "From and after July 1, 1978, the Kansas State Board of Healing Arts shall require every licensee to submit with the renewal application evidence of satisfactory completion of a program of continuing education required by the Board." Physicians not in the active practice of the healing arts in the State of Kansas may be exempted from the continuing medical education requirements for relicensure by signing an affidavit furnished by the Kansas State Board of Healing Arts. The following is a description of the CME requirements established by the Board, and an explanation of how to comply with the requirements.

As a basic requirement, the Board adopted the CME requirements of the Kansas Medical Society, which is the attainment of the American Medical Association "Physician's Recognition Award." The basic criteria for the PRA is 150 hours of educational experience over a three-year period. The educational activities are described in six separate categories. Below is a brief description of each category and the maximum number of hours that can be obtained in each category:

	Credit Hours Limit
Category 1 — CME activities with accredited sponsorship (60 hours required)	no limit
Category 2 — CME activities with non-accredited sponsorship	45 hrs.
Category 3 — Medical teaching	45 hrs.
Category 4 — Papers, publications, books, exhibits	40 hrs.
Category 5 — Non-supervised individual CME activities	45 hrs.
Category 6 — Other meritorious learning experiences	45 hrs.

Completion of AMA-PRA satisfies the requirement established by the Board. Because relicensure is on a yearly basis, and the PRA is on a three-year basis, a physician need only have a valid PRA in effect to be in compliance with the requirements of the Board.

Additionally, completion of the CME certification programs of any of the following organizations also constitutes compliance with the requirements of the Board:

American Academy of Dermatology
American Association of Neurological Surgeons
American College of Emergency Physicians
American College of Radiology
American Society of Colon and Rectal Surgeons
American Academy of Family Physicians
American College of Obstetricians and Gynecologists
College of American Pathologists/American Society of Clinical Pathologists
American Psychiatric Association
Any other specialty organization CME certification programs subsequently recognized by the AMA.

If you satisfy the requirements of any of the above organizations, you need *not* apply for the "Physician's Recognition Award." Each specialty organization will notify the Kansas Medical Society of your certification, and the Kansas Medical Society will in turn notify the Board that you have satisfactorily completed your requirements.

If you do not qualify for the specialty organization programs listed above, you must apply directly to the AMA for the "Physician's Recognition Award." Upon receipt of the award, the AMA will notify the Kansas Medical Society that you have completed the requirement.

Questions concerning CME requirement can be directed to the Board or the Kansas Medical Society. The addresses of each are as follows:

The Kansas State Board of Healing Arts
503 Kansas Avenue
Topeka, KS 66603
Telephone: (913) 296-7413

The Kansas Medical Society
1300 Topeka Boulevard
Topeka, KS 66612
Telephone: (913) 235-2383

Each individual physician is responsible for recording his own hours of attendance at postgraduate courses. The credit hours should be recorded on the AMA Physician's Recognition Award application form when 150 hours are accumulated. The application form should then be mailed directly to the AMA, 535 North Dearborn, Chicago, IL 60610.



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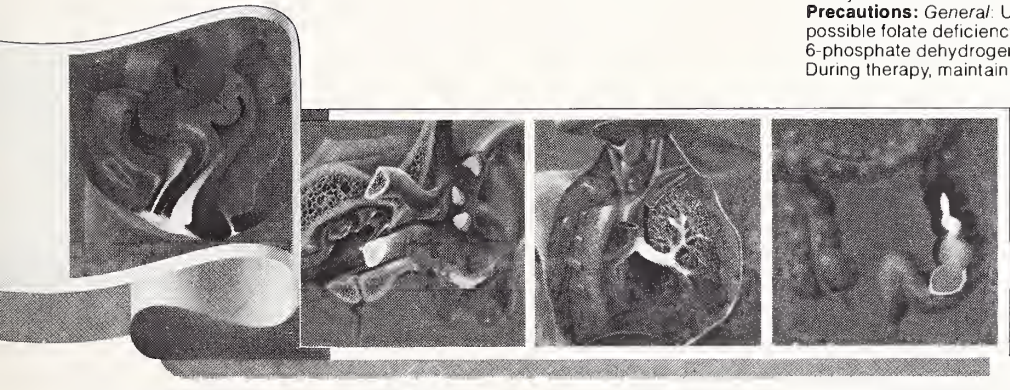


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a continuing record of high clinical effectiveness against common uropathogens

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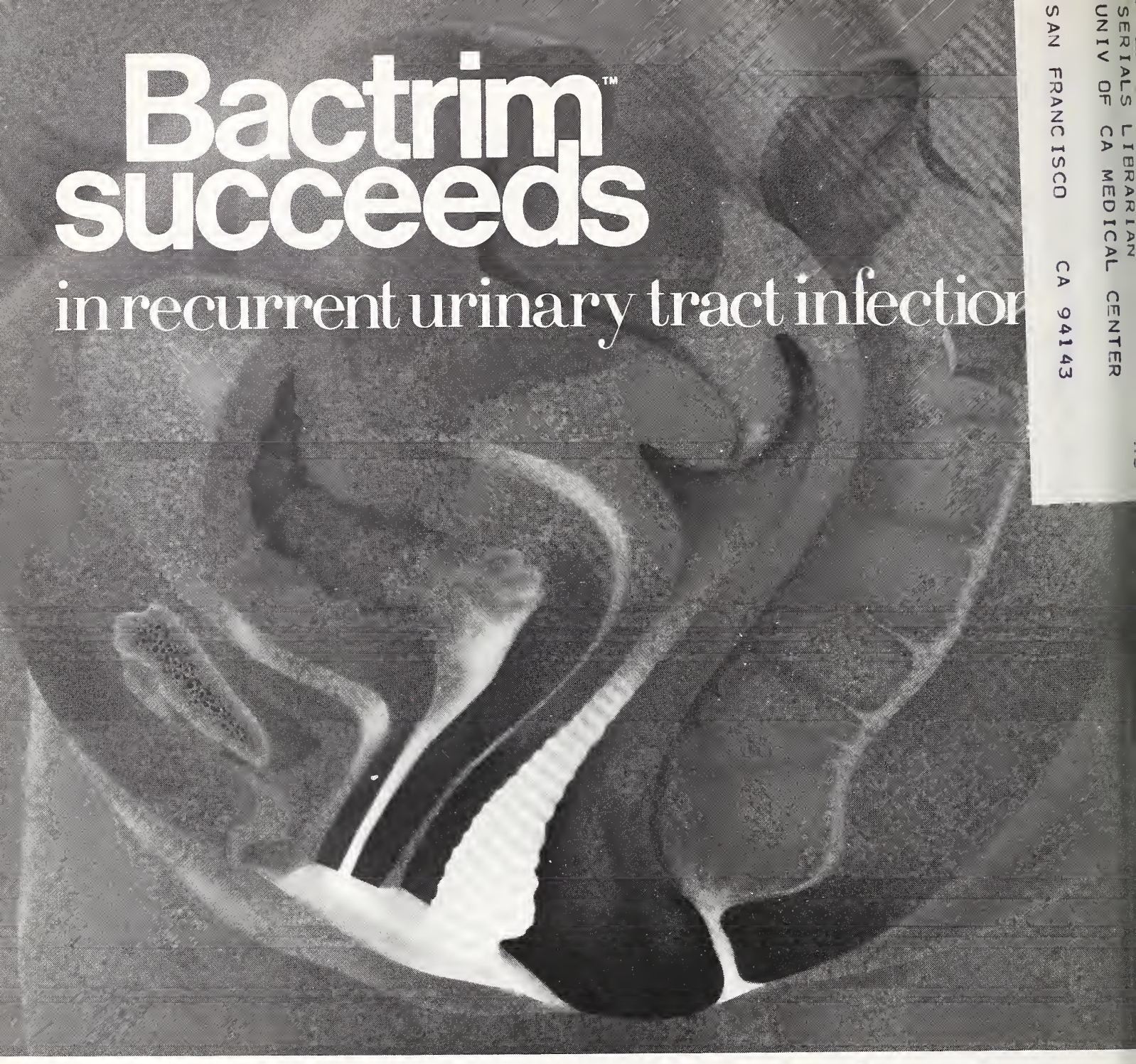
BACTRIM™ (trimethoprim and sulfamethoxazole/Roche)
Before prescribing, please consult complete product information, a summary of which follows:
Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination. *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.
For acute otitis media in children due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over other antimicrobials. To date, there are limited data on the safety of repeated use of Bactrim in children under two years of age. Bactrim is not indicated for prophylactic or prolonged administration in otitis media at any age.
For acute exacerbations of chronic bronchitis in adults due to susceptible strains of *Haemophilus influenzae* or *Streptococcus pneumoniae* when in physician's judgment it offers an advantage over a single antimicrobial agent.
For enteritis due to susceptible strains of *Shigella flexneri* and *Shigella sonnei* when antibacterial therapy is indicated.
Also for the treatment of documented *Pneumocystis carinii* pneumonitis.
Contraindications: Hypersensitivity to trimethoprim or sulfonamides; patients with documented megaloblastic anemia due to folate deficiency; pregnancy at term; nursing mothers because sulfonamides are excreted in human milk and may cause kernicterus; infants less than 2 months of age.
Warnings: BACTRIM SHOULD NOT BE USED TO TREAT STREPTOCOCCAL PHARYNGITIS. Clinical studies show that patients with group A β-hemolytic streptococcal tonsillopharyngitis have higher incidence of bacteriologic failure when treated with Bactrim than do those treated with penicillin. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.
Precautions: *General:* Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.
Bactrim may prolong prothrombin time in those receiving warfarin; reassess coagulation time when administering Bactrim to these patients.
Pregnancy: Teratogenic Effects: Pregnancy Category C. Because trimethoprim and sulfamethoxazole may interfere with folic acid metabolism, use during pregnancy only if potential benefits justify the potential risk to the fetus.
Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea, pseudomembranous colitis and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L.E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.
Dosage: Not recommended for infants less than two months of age.
URINARY TRACT INFECTIONS AND SHIGELLOSIS IN ADULTS AND CHILDREN, AND ACUTE OTITIS MEDIA IN CHILDREN:
Adults: Usual adult dosage for urinary tract infections—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days. Use identical daily dosage for 5 days for shigellosis.
Children: Recommended dosage for children with urinary tract infections or acute otitis media—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. Use identical daily dosage for 5 days for shigellosis.
For patients with renal impairment: Use recommended dosage regimen when creatinine clearance is above 30 ml/min. If creatinine clearance is between 15 and 30 ml/min, use one-half the usual regimen. Bactrim is not recommended if creatinine clearance is below 15 ml/min.
ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS IN ADULTS:
Usual adult dosage: 1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 14 days.
PNEUMOCYSTIS CARINII PNEUMONITIS:
Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.
Supplied: *Double Strength (DS) tablets*, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100; Prescription Paks of 20 and 28. *Tablets*, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40. *Pediatric Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); cherry flavored—bottles of 100 ml and 16 oz (1 pint). *Suspension*, containing 40 mg trimethoprim and 200 mg sulfamethoxazole per teaspoonful (5 ml); fruit-licorice flavored—bottles of 16 oz (1 pint).

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1. Rubin RH, Swartz MN: *N Engl J Med 303*.426-432, Aug 21, 1980. **2.** Data on file, Medical Department, Hoffmann-La Roche Inc.

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CONSTITUTION & BYLAWS

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CONSTITUTION

and

BY-LAWS

Kansas Medical Society

May 1982

ACT OF INCORPORATION

An Act to Incorporate the Kansas Medical Society

Be it enacted by the Governor and Legislative Assembly of the Territory of Kansas:

Section 1

Amory Hunting, S. B. Prentiss, J. P. Root, A. Fuller, C. F. Kobb, J. W. Robinson, J. B. Wheeler, L. C. Tolles, S. C. Harrington, A. Danford, C. E. Minor, J. B. Woodward, W. Madison, J. H. Phelps, O. Brown, Charles Robinson, M. F. Holladay, H. J. Canniff, A. J. Ritchie, M. Baily, J. M. Pelot, H. H. Beals, J. G. Blunt, T. Linsey, G. W. Beaumont, J. Leigh, A. Newman, H. Harttmann, Wm. Graham, and their associates and successors, who shall be elected to membership as hereinafter provided, are hereby constituted a body corporate and politic by the name of the Kansas Medical Society, and shall have perpetual succession forever. Said Society may have a common seal, and change or alter the same at pleasure.

Section 2

That members of said Society, in their corporate capacity, may elect such officers as they shall judge necessary for its government and the management of its affairs, determine the name, power, duty and term of office of each, also the time and manner of said elections.

Section 3

Said Society, by and in their corporate name, may have all the rights, privileges, and powers of a natural person in law and equity.

Section 4

Said Society may elect such persons to membership as they shall judge proper, and shall have power to expel, suspend, or disfranchise the same, as members, from all the rights and privileges of the Society; but such expulsion, suspension, or disfranchisement shall be by a vote of two-thirds of all the members present at a regular meeting of said Society, of which due notice shall have been given.

Section 5

Said Society shall have full power to make and enforce by-laws, and impose and collect at law any reasonable fines, not exceeding fifty dollars, as may be provided in said by-laws, for any and every violation or infraction thereof.

Section 6

Said Society shall issue certificates of membership to all its members, under such regulations as its by-laws may prescribe, and may also grant

(Continued on page 525)

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The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

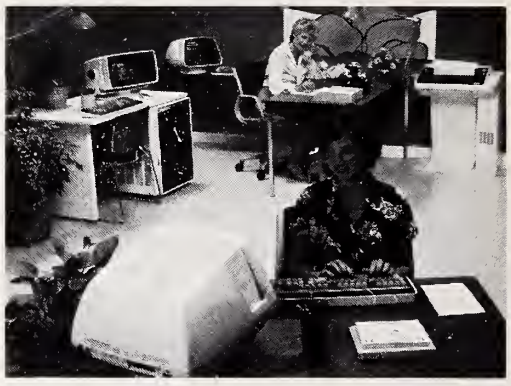
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
How Supplied: For oral administration, Valium scored tablets—2 mg, white; 5 mg, yellow; 10 mg, blue—bottles of 100* and 500;* Prescription Paks of 50, available in trays of 10.* Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25,† and in boxes containing 10 strips of 10.†

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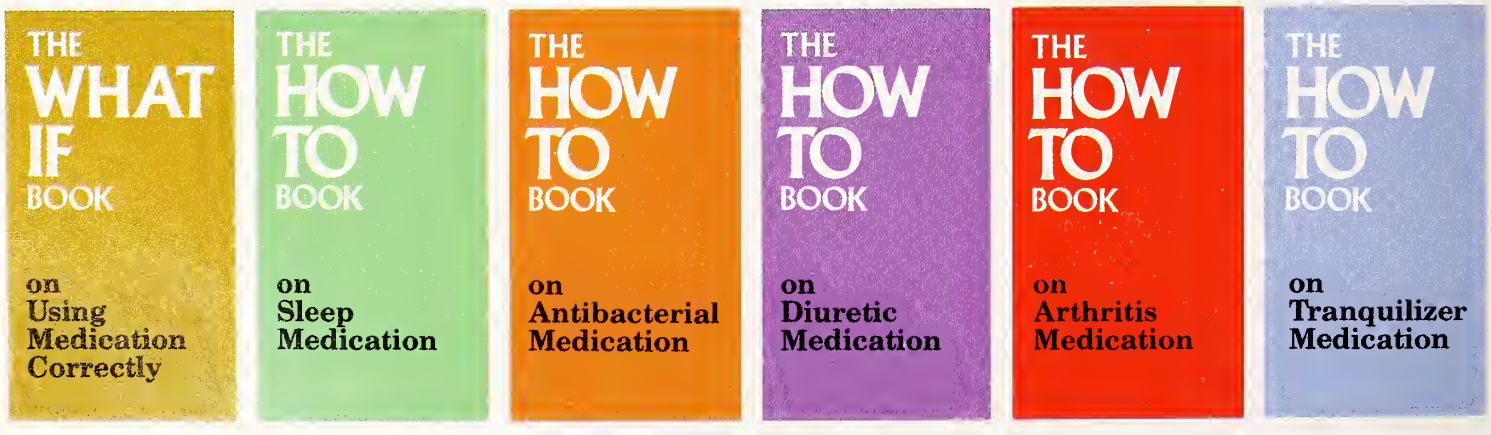
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KMS Constitution & By-Laws

(Continued from page 521)

licenses to all respectable physicians, non-graduates, who shall, on examination, be found qualified for the practice of medicine and surgery, or either, to practice those branches for which they are found qualified.

Section 7

Any three members of said Society may organize county or auxiliary societies in any county of this Territory; and said auxiliary society, when so organized, shall have all the powers and privileges, in the corporate name which they may adopt, that are conferred by this act upon the Kansas Medical Society; and the officers of said auxiliary societies shall be honorary members of the Kansas Medical Society.

Section 8

A meeting of the corporators, or a part thereof, shall be held in Lawrence, on February 10, A.D., 1859, for the purpose of electing the first officers and completing the organization.

Section 9

This act to take effect and be in force from and after its passage.

C. W. Babcock
President of the Council
Approved February 10, 1859

A. Larzelere
Speaker of the House of Representatives
S. Medary, Governor

CHARTER DECLARED VALID 1881

In 1880 a lawsuit reached the Supreme Court of Kansas contending in part:

"1. That the charter of the Society has expired by statutory limitation;

"2. That the power of the territorial legislature, being permissive and temporary only, could confer no invested right, by contract or otherwise, which would bind the state against its consent;

"3. That the charter of the Society was granted by a territorial act, not accepted or preserved by the state; and

"4. That the legislature did not and has not the power under the constitution to recognize or validate the existence of the Society, nor to grant it additional powers by the act of 1879. . . ."

In the January term, 1881, in the case, STATE OF KANSAS ex rel. v. D. W. Stormont and others, the Supreme Court of the State of Kansas, with all justices concurring, said in part:

"The Society was incorporated, by a special act of the territorial legislature, on the 10th day of February 1859. . . ." *It is,* ". . . hereby constituted a body corporate and politic, by the name of the 'Kansas Medical Society,' and shall have perpetual succession forever. It is conceded that the legislature of the territory had the power to incorporate the Society by a special act. Having the power to create the corporation, it had the further power to endow it with all the attributes of a corporation, not inconsistent with the provisions of the Constitution of the United States, and the act organizing the territory of Kansas, approved May 30, 1854. . . . Therefore, within this definition, immortality is a legitimate attribute to be conferred on a corporation. . . . When not limited or forbidden by constitutional or organic law, the right to confer perpetual succession by legislative authority, so far at least as human agency can confer such an attribute, cannot be logically questioned. . . . This much is clear: The charter did not expire by the law of 1855.

"Our conclusions upon the foregoing matters are that the Kansas Medical Society was lawfully chartered by the territorial legislature; that it was legally endowed with perpetual succession forever; that the constitution did not suspend or repeal its charter; that, if the state legislature has the power to suspend or repeal the charter (which we do not decide) it has never exercised, or attempted to exercise, the power; and that the Society is a lawfully existing corporation."

CONSTITUTION

ARTICLE I — Title and Definition

The name of this organization is THE KANSAS MEDICAL SOCIETY. The SOCIETY is comprised of the Component Societies chartered by this organization.

ARTICLE II — Objects

The object of this SOCIETY is to unite the medical profession of the State of Kansas in promoting the science and art of medicine and protecting the health of the citizens of this State.

ARTICLE III — Component Societies

County or multi-county societies holding a charter from this organization are known as Component Societies.

ARTICLE IV — Members

The KANSAS MEDICAL SOCIETY is composed of members of the Component Societies and others as provided in the By-Laws.

ARTICLE V — House of Delegates

The House of Delegates is the primary legislative and governing body of this SOCIETY. The members of the House of Delegates will be elected by the Component Societies as provided in the By-Laws. This body will transact the business of the SOCIETY and will elect officers except as otherwise provided in the By-Laws.

ARTICLE VI — Officers

The officers of this SOCIETY are a President, a President Elect, a First Vice President, a Second Vice President, a Secretary, a Treasurer, a Speaker and a Vice Speaker of the House, Delegates and Alternate Delegates to the American Medical Association. The terms of office, qualifications, and method of election shall be provided in the By-Laws.

ARTICLE VII — Council Districts and the Council

The boundaries of the Council Districts shall be specified in the By-Laws. The Council consists of one Councilor from each Council District, the officers of the SOCIETY, and advisory members as designated in the By-Laws. The Council may transact business of the SOCIETY between sessions of the House of Delegates subject to the approval of that body and as prescribed in the By-Laws.

ARTICLE VIII — Meetings

The SOCIETY will hold an annual meeting for the presentation and discussion of subjects pertaining to the science and art of medicine. The House of Delegates shall convene at the annual meeting and at other times as necessary for the transaction of the business of the SOCIETY. The place of the annual meeting shall be approved by the House of Delegates, following a recommendation of the Council.

ARTICLE IX — Funds, Dues, Assessments

Funds for the functioning of this SOCIETY shall be raised by an equal annual dues or by assessment of the Members who are subject to these charges as provided in the By-Laws. The amount of dues and assessments shall be determined by the Council and approved by the House of Delegates.

ARTICLE X — Seal

The following insignia shall be the official seal of this SOCIETY:



The official seal shall at all times remain in the custody of the Secretary.

Amendments to Constitution

ARTICLE XI — Amendments

Amendments to this Constitution require an affirmative vote of two-thirds of the Delegates present provided the question has been introduced at the previous annual session, or upon recommendation by the Council and published twice in *The Journal of the Kansas Medical Society,* or submitted by the Council to each Component Society at least two (2) months in advance of the meeting.

BY-LAWS

1.0 MEMBERSHIP

1.1 Each component society shall judge the qualifications and classify its own members. When names are submitted on a properly prepared roster to the state office along with payment of the full annual assessment to the Kansas Medical Society, if required by their classification, the listed physicians shall become members of the Kansas Medical Society, and they shall remain so as long as they are members in good standing in their component societies and the dues to the Kansas Medical Society remain paid, provided that

1.11 Every active member of the Society shall fulfill the requirements of postgraduate medical education set forth by the Commission for Education of the Kansas Medical Society.

1.111 Out of State members shall be exempt from the continuing medical education requirement.

1.2 Before a charter is issued to any component society, ample and full opportunity to become a member shall be given to every physician residing or practicing in the area encompassed by that society.

1.3 Every eligible physician shall be privileged to apply for membership in an established component society society encompassing his area of practice.

1.4 When a member's practice is interrupted by an authorized absence, his dues will not be refunded except upon recommendation by the component society to which he belongs.

1.5 Members may attend an annual session after first registering and being verified.

1.6 Classification of members

1.61 Members with the right to vote and hold office:

1.611 Members who pay full dues. Members of a component society who hold a degree of Doctor of Medicine, Doctor of Osteopathy, or their equivalent, and are fully licensed to practice medicine and surgery by the Kansas State Board of Healing Arts.

1.6111 Active Members (1st year) — Physicians in their first year of full time medical practice after completing their residency requirements. They shall pay 50 per cent of regular dues and assessments the first full dues year.

1.612 Dues-Exempt Members: Component societies may designate members to be excused from paying dues in these categories:

1.6121 Personal Exemption: Members for whom extended illness or financial limitations create genuine difficulty in paying the dues.

1.6122 Retirement: Members who have retired from active practice. Members in this category may hold an inactive license to practice medicine and surgery.

1.6123 Service: Members temporarily serving with the armed forces except as provided in 1.7.

1.6124 Emeritus: Members over seventy (70) years of age, with dues-paying status for ten (10) years or more may apply for this category, or elect to retain active membership.

1.6125 Probationary Members: Upon certification by the secretary of a component society that a member has been accepted on a probationary basis, the Kansas Medical Society will place his name on the membership rolls. The probationary member will receive the *Journal* and all other correspondence forwarded from the state office. He may serve on committees and commissions but is not eligible to serve as a state or AMA delegate, councilor, or officer. One-half regular dues will be assessed during this period. This category of membership terminates when the component society either votes him into active membership or drops him from their membership rolls.

1.6126 Interns and Residents engaged in full-time training shall be given full privileges of membership including the right to vote and hold office and be assessed annual dues of ten dollars ($10.00).

1.6127 Medical Students shall be assessed no dues and shall receive the *Journal*.

1.62 Members with full privileges except for the right to vote and hold office. They apply for membership through a component society but are assessed less than the full amount of dues:

1.622 Academic Members: Physicians whose activities are predominantly teaching, administration, or research in approved medical schools. Those with a teaching classification below the level of assistant professor shall pay fifty per cent (50%) of

the regular dues and assessments.

1.623 Associate Members: Physicians who are active members of another state medical society and wish to affiliate with the Kansas Medical Society. They shall pay fifty per cent (50%) of the regular dues and assessments.

1.63 Honorary Members: Persons outside the membership of this Society may be designated Honorary Members by a majority vote of the House of Delegates. They pay no dues and may not vote or hold office:

1.631 Members of the medical societies of other states or of foreign medical societies recognized by the American Medical Association.

1.632 Physicians from Kansas, having graduated from an accredited school of medicine, and who are serving outside the United States as missionaries or in education or philanthropic work.

1.633 Non-physicians, whether Kansans or not, who have made an outstanding contribution to the Kansas Medical Society or to the field of Medicine in general.

1.634 Physicians from the membership of this Society are named for notable achievement in the field of medicine or extraordinary service in the interest of this Society. They are granted Honorary status by a vote of the House of Delegates and pay no dues, but if they were previously voting members, retain the right to vote and hold office.

1.7 Leave of Absence: Leave of absence is granted any member for the period specified by the secretary of his component society, in written certification to the secretary of this Society, excepting that an absence shorter than six (6) months will not alter his previous status. A member on leave of absence for more than six (6) months is exempt from payment of dues in any full six (6) months of absence calculated from 1 January and 1 July, upon certification by his component society.

1.71 Dues will not be exacted from the member on leave at the time they become payable. If he is absent for a full year or more, the prepaid dues will be applied to the year of his return.

1.72 A leave of absence exceeding one year, calculated from the first certification in the annual report of membership, must be recertified in each subsequent annual report, or until notification of reinstatement (or discontinued membership) is received from the component society.

1.8 Dues Refund: Refunds of one half (½) membership dues will be considered on an individual basis upon application to the Executive Committee prior to July 1.

1.81 One-half Year Memberships: One-half year memberships will be considered on an individual basis upon application to the Executive Committee.

2.0 ASSESSMENTS

2.1 The amount of the annual assessment for the Kansas Medical Society only shall be determined by the Council after consideration of the annual budget for the ensuing year and announced to the various component societies not less than sixty (60) days before the beginning of each fiscal year. Such assessments shall be levied against and paid by the component societies in the manner provided by this Constitution and By-Laws, except that any new member of this Society being accepted by a component society after July 1, shall be assessed one-half the sum decided upon by the Council, and shall be accorded all the rights and benefits of this Society, including defense, until the succeeding January 1.

2.2 Assessments shall include subscription to the *Journal of the Kansas Medical Society* at a rate determined by the Editorial Board with the approval of the Council.

2.3 Special assessments may be levied as needed. Request for special assessments will be referred to the Council for study, whereupon the Council shall make a recommendation to the House of Delegates for final action. All special assessments are mandatory upon all full dues-paying members unless otherwise specified.

3.0 ANNUAL AND SPECIAL SESSIONS

3.1 The Society shall hold an annual session at the time and place determined by the Council at the preceding annual session. The date and location of the annual session may be changed by the President subject to the approval of the Council. *Provided:* that each component society is notified of the change at least five (5) weeks before the new date is selected.

3.2 Special meetings of the Society may be called by the President upon approval of the Council.

4.0 GENERAL MEETINGS AND SECTIONS

4.1 The Scientific Assembly at the annual sessions may be divided into general meetings and sections by the local Committee on Arrangements with approval of the Committee on State Meeting Format.

4.2 The general meetings and sections of the Society shall be devoted to scientific work together with such reports and announcements as may come from the House of Delegates or the Council.


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Comprehensive decongesting, antihistaminic and anti-secretory reliever for patients with nasal, sinus and other upper respiratory irritation.

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• Blocks the cough • Loosens mucus
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RU-TUSS®

Tablets

DESCRIPTION

Each prolonged action tablet contains:

Phenylephrine Hydrochloride	25 mg
Phenylpropanolamine Hydrochloride	50 mg
Chlorpheniromine Maleate	8 mg
Hyoscyamine Sulfate	0.19 mg
Atropine Sulfate	0.04 mg
Scopolamine Hydrobromide	0.01 mg

Ru-Tuss Tablets act continuously for 10 to 12 hours.

Ru-Tuss Tablets are an oral antihistaminic, nasal decongestant and anti-secretory preparation.

INDICATIONS AND USAGE Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues. Phenylephrine and phenylpropanolamine combine to exert a vasoconstrictive and decongestive action while chlorpheniramine maleate decreases the symptoms of watering eyes, post nasal drip and sneezing which may be associated with an allergic-like response. The belladonna alkaloids, hyoscyamine, atropine and scopolamine further augment the anti-secretory activity of Ru-Tuss Tablets.

CONTRAINDICATIONS Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.

WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.

PRECAUTIONS Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with glaucoma, or urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See Warnings).

OVERDOSAGE Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.

ADVERSE REACTIONS Hypersensitivity reactions such as rash, urticaria, leukopenia, agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerotomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.

DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.

HOW SUPPLIED:

Bottles of 100 Tablets	NDC 0524-0058-01
Bottles of 500 Tablets	NDC 0524-0058-05

Federal law prohibits dispensing without prescription.

DISTRIBUTED BY:
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Shreveport, Louisiana 71106

MANUFACTURED BY:
Vitarine Company, Inc.
Springfield Gardens, New York 11413

RU-TUSS®

Expectorant

DESCRIPTION

Each fluid ounce of Ru-Tuss Expectorant contains:

Codeine Phosphate	65.8 mg
(WARNING: MAY BE HABIT FORMING)	
Phenylephrine Hydrochloride	30 mg
Phenylproponolomine Hydrochloride	20 mg
Pheniramine Maleate	20 mg
Pyrilamine Maleate	20 mg
Ammonium Chloride	200 mg
Alcohol	5%

Ru-Tuss Expectorant is an oral antitussive, antihistaminic, nasal decongestant and expectorant preparation.

INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief of upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms associated with hay fever, allergies, nasal congestion and cough due to the common cold.

CONTRAINDICATIONS Hypersensitivity to antihistamines. Concomitant use of an antihypertensive or antidepressant drug containing a monoamine oxidase inhibitor is contraindicated.

Ru-Tuss Expectorant is contraindicated in patients with glaucoma, bronchial asthma and in women who are pregnant.

WARNINGS Ru-Tuss Expectorant contains codeine phosphate, therefore, the patient should be warned of the potential that this drug may be habit forming. Ru-Tuss Expectorant may cause drowsiness. Patients should be warned of the possible additive effect caused by taking antihistamines with alcohol, hypnotics, sedatives and tranquilizers.

PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicle or operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease.

Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.

ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions.

DOSAGE AND ADMINISTRATION Adults: 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.

Children 6 to 12 years of age: ½ the adult dose, not to exceed 6 teaspoonfuls in any 24-hour period. Children 2 to 6 years of age: ½ teaspoonful every 4 hours, not no exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age: Use as directed by a physician.

HOW SUPPLIED: (16 fl. oz.)

Pint Bottles	NDC 0524-1010-16

Federal law prohibits dispensing without prescription.

MANUFACTURED AND DISTRIBUTED BY:
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Shreveport, Louisiana 71106

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Shreveport, Louisiana 71106
Pioneers in medicine for the family

4.3 The Society reserves the right to publish in the *Journal* any paper read before the Society. The Society also shall be entitled to a copy of the paper if so desired.

4.4 Registration for General and Sectional Meetings. Only the following shall be permitted to register and attend:

4.41 Members: Members of the Society who are in good standing.

4.42 Invited Guests: Non-members of the Society may be invited to the general session or sections, but may be excluded by the Executive Committee of the Society or officers of the Sections.

4.43 Residents and Interns: Residents and interns who are graduates of approved medical or osteopathic schools and who are certified by their hospital.

4.44 Medical Students: Medical students of approved medical and osteopathic schools who are certified by their schools.

4.5 The Scientific Assembly

The Scientific Assembly may be divided into sections representing the various specialties in the practice of medicine. Any member of the Society shall be entitled to attend the scientific programs of specialty sections.

4.51 An application for a new section or a request to discontinue a section shall be referred to the Council for study, whereupon the Council shall make a recommendation to the House of Delegates for final action.

4.52 Sections will be governed by their respective officers.

4.53 Officers will consist of a chairman, vice-chairman and secretary-treasurer.

4.54 Terms of office will be for one (1) year and each officer may succeed himself.

4.55 Each section will prepare its own program subject to approval by the Committee on State Meeting Format. Programs must be submitted to the committee far enough in advance to be included in the annual session program.

4.56 Papers and records of proceedings of the sections shall become the property of the Society and filed in the central office of the Society.

4.57 All resolutions and memorials of a general meeting or a section, intended to be issued in the name of the Society, must be approved by the Council or House of Delegates before being issued or becoming effective.

4.58 Each section may enact its own rules of conduct that do not conflict with the Constitution or By-Laws of the Society. Sections approved by the Society include:

4.581 Kansas Society of Anesthesiology

4.582 Section on Ophthalmology

4.583 Kansas Chapter, American Society of Internal Medicine

4.584 Kansas Obstetrical Society

4.585 Kansas Orthopedic Society

4.586 Kansas Society of Pathologists

4.587 Kansas Chapter, American Academy of Pediatrics

4.588 Kansas Psychiatric Society

4.589 Kansas Chapter, American College of Radiology

4.5810 Kansas Chapter, American College of Surgeons

4.5811 Kansas Chapter, American Academy of Family Physicians

4.5812 Kansas Allergy Society

4.5813 Kansas Urological Society

4.5814 Section on Ear, Nose and Throat

4.5815 Kansas Dermatological Society

4.5816 Kansas Neurosurgical Society

4.5817 The Kansas Neurological Society

4.5818 Section on Nuclear Medicine

4.5819 Kansas Chapter, American College of Emergency Room Physicians

4.5820 Resident Physicians Society

5.0 HOUSE OF DELEGATES

5.01 Introduction and General Powers

The House shall consider and advise on matters of interest to the medical profession and of the public in those important matters wherein it is dependent upon the medical profession and shall advocate all proper medical and health legislation, and the diffusion of popular information in relation thereto.

It shall attend to the scientific work and spirit of this Society, and it shall constantly attempt to improve the quality of health care in this state.

The House of Delegates is the ultimate authority in legislative and business affairs of this Society. It may appoint committees from its own members or from members of the Society. Such committees shall report to the House, and may be present and participate in the debates on their reports.

The House may delegate and empower the Council or a special standing committee to employ, advise and supervise an Executive Director whose duties shall be as provided in these By-Laws.

The House shall review the annual budget submitted by the Council and shall make annual appropriations for the expenditures of this Society. It may amend current appropriations at any meeting.

A summary of the proceedings of the House of Delegates shall be published following each annual session in the *Journal of the Kansas Medical Society*.

5.1 Composition

The House of Delegates shall be composed of delegates selected by the component societies, by the elected councilors and in their absence, their alternates, by the recognized sections, the past presidents, and the elected officers of this Society. Members of the Society may attend plenary sessions and others may attend upon invitation of the Speaker.

5.2 Apportionment

5.21 Component Societies: Each component society having made its annual report and paid its assessments as provided in this Constitution and By-Laws shall elect one (1) delegate and one (1) alternate to the House of Delegates for each twenty (20) members and major fraction thereof. Each component single county society shall be entitled to at least one (1) delegate and one (1) alternate, and provided that component multicounty societies have fewer than seventy-five (75) members are entitled to elect one (1) delegate and one (1) alternate, *plus* one (1) delegate and one (1) alternate for each ten (10) members and major fraction.

Student and resident members shall not be counted for the purposes of determining the number of delegates.

5.22 Organized Specialties: Each organized specialty section recognized by the Society shall be entitled to one (1) delegate and one (1) alternate to be chosen by the section.

5.23 Listing Names: It is the duty of the secretary of each component society and specialty section to send the names of the delegate(s) and alternate(s) to the Executive Director at least sixty (60) days prior to each session.

5.24 Each district councilor shall be a member of the House of Delegates during his Council tenure and in addition to the authorized number of delegates from his component society.

5.3 Delegates

5.31 Qualifications

5.311 Delegates: Each delegate must be a member in good standing of this Society.

5.312 Alternates: An alternate to each delegate shall be elected to substitute for that delegate. Each alternate must be a member in good standing of the Society.

5.313 Substitute Alternate: The House may elect by majority vote a substitute alternate to serve as a delegate for that meeting for a component society which has no delegate or alternate present.

5.314 Proxies: No proxies are recognized.

5.32 Term

5.321 Length: Each delegate and alternate is elected for one (1) year and shall assume office at the first session of the House succeeding his selection.

5.322 Vote: Each delegate is entitled to one (1) vote.

5.33 Registration

All persons attending a session of the House shall register prior to their admission. These registrations are certified by the Committee on Credentials.

5.4 Procedure

5.41 Order of Business

5.411 First Session: The order of business of the first session of the House, unless otherwise ordered by a two-thirds vote of the delegates present, shall be:

5.4111 Registration of delegates, Society members and visitors.

5.4112 Call to order by the Speaker.

5.4113 Announcement of number of delegates present and registered and the presence of an official quorum.

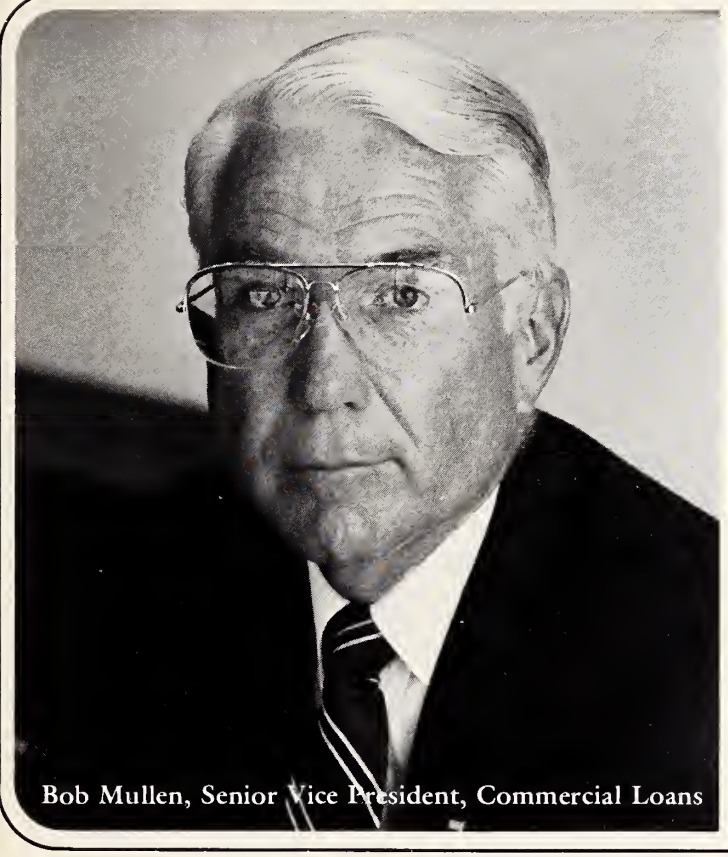
5.4114 Reading of the minutes of last and any special meeting.

5.4115 The report of the nominating committee is read, after which nominations are made from the floor for the office of President Elect, First Vice President, Second Vice President, Speaker, Vice Speaker, Secretary, Treasurer, AMA Delegate and AMA Alternate Delegate, and a ballot vote where three or more candidates have been nominated for one office so that the election at the second session of the House shall present not more than two (2) candidates for each office.

5.4116 Report of the Constitutional Secretary shall be distributed to all persons in attendance. The Constitutional Secretary shall call attention of the House to any statistics relating to membership that are of exceptional importance.

5.4117 The report of the Treasurer, including a proposed budget for the coming year, shall be distributed to all persons present. The Treasurer shall call attention to any specific financial items that are exceptional. The House shall immediately thereafter

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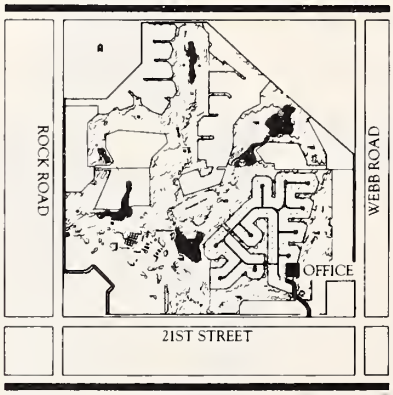
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act to approve the report of the Treasurer.

5.4118 The report of the Editor of the *Journal* shall be prepared and distributed to all persons present.

5.4119 The Speaker of the House of Delegates may invite such persons to present written or oral reports to the House of Delegates which in his judgment will be important for information to the House.

5.41110 The report of the Executive Director shall be prepared and distributed to all persons present.

5.41111 Unfinished business.

5.41112 Reports and presentation of resolutions from Commissions.

5.41113 New business and the introduction of resolutions from component societies, specialty societies, and delegates.

5.41114 Announcements — to include: (1) members of each reference committee; (2) time and place of reference committee meetings; (3) names and districts of expiring councilor and alternate terms; (4) results of primary election; (5) other.

5.41115 Adjournment to reconvene at second meeting.

5.412 Section Session: The official order of business for the second meeting of the House of each annual session shall be:

5.4121 Registration and seating of delegates, Society members and visitors.

5.4122 Call to order by the Speaker.

5.4123 Announcement of number of delegates, the presence of an official quorum.

5.4124 Election of officers (by ballot)

5.4125 Report of reference committees.

5.4126 Unfinished business.

5.4127 New business (this requires consent of a two-thirds majority of delegates present).

5.4128 The Speaker announces the President Elect is now President. The President is invited to present to the House an outline of his plans for the coming year at the time he assumes office. The President then announces the councilors and alternates elected and meeting place of the Council.

5.4129 Installation by the President of the new Speaker and Vice Speaker.

5.41210 Adjournment

5.413 Special Session: The official order of business for a special session of the House shall be:

5.4131 Registration of delegates, Society members and visitors.

5.4132 Call to order by the Speaker.

5.4133 Announcement of number of delegates present and the presence of a quorum.

5.4134 Announcement of business for which session was convened.

5.4135 Report of the commission chairmen and officers pertinent to the subjects for which the special session is convened.

5.4136 Assignment of resolutions to reference committees.

5.4137 Adjournment of the First Session.

5.4138 Second Session — Call to order by the Speaker.

5.4139 Report of the Reference committees.

5.41310 Announcements.

5.41311 Adjournment.

5.42 Privilege of the Floor

The House of Delegates by a two-thirds vote of those present may invite any person to address the House.

5.43 Introduction of Resolutions:

Reports and resolutions for consideration of the House shall be sent to the Executive Office at least six (6) weeks before the date of the annual session. Resolutions or amendments introduced on the floor of the House must be submitted to the Speaker in writing at the time of introduction.

5.44 Process of Resolutions:

5.441 First Session

5.4411 At the first session of the House, all business shall be introduced by title, and referred without debate or action to a reference committee, except as otherwise stipulated in these By-Laws.

5.4412 Resolutions not previously published and distributed to the members of the House shall be read in full.

5.4413 As each resolution is introduced, the Speaker shall designate it with a number.

5.442 Second Session

5.4421 At the second session, the House will receive the report of each reference committee on all business referred to it.

5.44211 If, in the opinion of the reference committee chairman, a resolution can be acted upon without additional discussion, he shall direct the resolution to be placed on the consent calendar. A list of such resolutions shall be presented at the end of the reference committee report. The Speaker shall ask if there is any objection. If an objection appears to any resolution, it shall be removed from the con-

HYPERTENSION:



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INDERAL exhibits few of the disturbing side effects of methyldopa and reserpine. Sedation, depression, and impotence are rare.* Tolerance is not likely to occur, as it frequently does with methyldopa. For the vast majority of patients—INDERAL means a step toward improving the quality of life. (INDERAL should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.)*

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*Please see following page for Brief Summary of Prescribing Information.



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BEFORE USING INDERAL (PROPRANOLOL HYDROCHLORIDE), THE PHYSICIAN SHOULD BE THOROUGHLY FAMILIAR WITH THE BASIC CONCEPT OF ADRENERGIC RECEPTORS (ALPHA AND BETA), AND THE PHARMACOLOGY OF THIS DRUG

CONTRAINDICATIONS

1) bronchial asthma; 2) allergic rhinitis during the pollen season; 3) sinus bradycardia and greater than first degree block; 4) cardiogenic shock; 5) right ventricular failure secondary to pulmonary hypertension; 6) congestive heart failure (see WARNINGS) unless it is secondary to a tachyarrhythmia treatable with propranolol; 7) in patients on adrenergic-augmenting psychotropic drugs (including MAO inhibitors), and during the two week withdrawal period from such drugs.

WARNINGS

CARDIAC FAILURE: In congestive heart failure, inhibition with beta-blockade carries the potential hazard of further depressing myocardial contractility and precipitating cardiac failure. In patients already receiving digitalis, propranolol may reduce the positive inotropic action of digitalis and may have an additive depressant effect on AV conduction.

IN PATIENTS WITHOUT A HISTORY OF CARDIAC FAILURE, in rare instances, cardiac failure has developed during propranolol therapy. At the first sign of impending cardiac failure, patients should be fully digitalized and/or given a diuretic, and observed closely: a) if cardiac failure continues, despite adequate digitalization and diuretic therapy, propranolol should be immediately withdrawn; b) if tachyarrhythmia is being controlled, patients should be maintained on combined therapy and closely followed until threat of cardiac failure is over.

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuation of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced and the patient carefully monitored. In addition, when INDERAL is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease, who are given propranolol for other indications.

IN PATIENTS WITH THYROTOXICOSIS, possible deleterious effects from long term use have not been adequately appraised. Give special consideration to propranolol's potential for aggravating congestive heart failure. Propranolol may mask the clinical signs of developing or continuing hyperthyroidism or complications and give a false impression of improvement. Propranolol should be withdrawn slowly, since abrupt withdrawal may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

IN PATIENTS UNDERGOING MAJOR SURGERY, beta-blockade impairs the ability of the heart to respond to reflex stimuli. Except in pheochromocytoma, propranolol should be withdrawn 48 hours prior to surgery. In case of emergency surgery, the effects of propranolol can be reversed by administration of beta-receptor agonists such as isoproterenol or levarterenol, but such patients may be subject to protracted severe hypotension. Difficulty in restarting and maintaining the heart beat has been reported.

IN PATIENTS PRONE TO NONALLERGIC BRONCHOSPASM (e.g., CHRONIC BRONCHITIS, EMPHYSEMA), administer with caution, since propranolol may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta-receptors.

DIABETICS AND PATIENTS SUBJECT TO HYPOGLYCEMIA: Propranolol may prevent the appearance of premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia, especially in patients with labile diabetes. A precipitous elevation of blood pressure may accompany hypoglycemic attacks.

USE IN PREGNANCY. Safe use in human pregnancy not established. Embryotoxic effects have been seen in animals at doses about 10 times the maximum recommended human dose.

PRECAUTIONS

Patients receiving catecholamine depleting drugs such as reserpine should be closely observed if propranolol is administered, since it may occasionally produce hypotension and/or marked bradycardia resulting in vertigo, syncopal attacks, or orthostatic hypotension.

Observe laboratory parameters at regular intervals. Use with caution in patients with impaired renal or hepatic function.

ADVERSE REACTIONS

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; arterial insufficiency, usually of the Raynaud type; thrombocytopenic purpura. *Central Nervous System:* lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. *Gastrointestinal:* nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis. *Allergic:* pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress. *Respiratory:* bronchospasm. *Hematologic:* agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura. *Miscellaneous:* reversible alopecia. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta-blocker (practolol) have not been conclusively associated with propranolol. *Clinical Laboratory Test Findings:* Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

HOW SUPPLIED

TABLETS

—Each hexagonal-shaped, orange, scored tablet is embossed with an "I" and imprinted with "INDERAL 10," contains 10 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0421-81) and 1,000 (NDC 0046-0421-91). Also in unit dose package of 100 (NDC 0046-0421-99).

—Each hexagonal-shaped, blue, scored tablet is embossed with an "I" and imprinted with "INDERAL 20," contains 20 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0422-81) and 1,000 (NDC 0046-0422-91). Also in unit dose package of 100 (NDC 0046-0422-99)

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—Each hexagonal-shaped, yellow, scored tablet is embossed with an "I" and imprinted with "INDERAL 80," contains 80 mg propranolol hydrochloride, in bottles of 100 (NDC 0046-0428-81) and 1,000 (NDC 0046-0428-91). Also in unit dose package of 100 (NDC 0046-0428-99).

The appearance of these tablets is a trademark of Ayerst Laboratories.

Store at room temperature (approximately 25° C).

INJECTABLE

—Each ml contains 1 mg of propranolol hydrochloride in Water for Injection. The pH is adjusted with citric acid. Supplied as 1 ml ampuls in boxes of 10 (NDC 0046-3265-10).

Store at room temperature (approximately 25° C).

7997/882

Reference: 1. Freis, E. D. Hypertension (Suppl. II) 3:230 (Nov.-Dec.) 1981.

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sent calendar and placed before the House for individual consideration. If no objection appears, the resolutions on the consent calendar will be placed as a package before the House for action.

5.4422 Each item of business so reported upon shall be subject to debate and amendment prior to final action.

5.4423 No new business may be considered at the second session of the House except as provided in the By-Laws.

5.4424 If a reference committee fails to submit a report at the second session of the House upon any resolution referred to it, such resolution may be placed before the House by the Speaker and must be so placed upon request of any member.

5.443 Special Session

Resolutions may be considered by the House acting as a whole or by a reference committee prior to action by the House.

5.444 Expiration

Official policies established through resolutions at the House of Delegates shall be in effect for a period of five (5) years, at which time that policy position will be reviewed by the Executive Committee and will expire subject to the approval by the House of Delegates unless superseded or continued by another resolution.

5.45 Quorum

Thirty-five per cent (35%) of constitutionally provided delegates or their respective alternates shall constitute a quorum of the House of Delegates at any regular or special meeting.

5.5 Sessions of the House of Delegates

5.51 Location: The time and place shall be determined by the Council. Notice of annual meetings shall be sent to each component society prior to January 1 of the year the meeting is to be held and shall state the time and place of the meeting.

5.52 Invitation From Component Societies: The Council shall, not later than October, consider invitations received from component societies to serve as host to the annual session for the fifth year following and shall select the meeting place and the date. Should invitations not be received, the Council shall select a meeting place.

5.53 Regular: The House of Delegates shall conduct two meetings, separated by at least twenty-four (24) hours, during each annual session. These may be adjourned and reconvened as necessary.

5.54 Special: Special sessions shall be called by the Speaker as requested by the President upon approval of the Council or the Executive Committee. Special sessions shall be called by the Speaker upon the written request of twenty-five per cent (25%) of the delegates from component societies. When a special meeting is called, the Executive Director shall notify each component society at least twenty (20) days before the meeting date. The notice shall specify the time and place of meeting and the purpose for which it is called.

5.6 Meetings

5.61 Plenary: The House may conduct a meeting to which any person may be admitted. By majority vote of the delegates present, a plenary meeting may become a closed or executive meeting.

5.62 Closed: A closed meeting is restricted to members of this Society and to members of the staff.

5.63 Executive: An executive meeting is limited to delegates and to such employees of this Society necessary for the functioning of the House.

5.7 Designation

The Credentials Committee and the Reference Committee are regularly appointed committees of the House of Delegates.

5.8 Membership

5.81 The Credentials Committee shall consist of three (3) members from separate Council districts appointed by the Speaker for a term of one (1) session. The Speaker shall designate the chairman.

5.82 Each Reference Committee shall consist of not less than three (3) nor more than seven (7) members of the House appointed by the Speaker. The Speaker shall designate the chairman.

5.9 Duties

5.91 Credentials Committee

5.911 The Credentials Committee shall examine the credentials of all who seek admission to the House, and rule on the seating of all delegates. Any member of the Society registered for the annual meeting may be admitted to the visitors' section.

5.912 An individual whose credentials are questioned may appeal to the House immediately following the report of the Credentials Committee that a quorum exists and the issue may be decided at that time.

5.913 The Speaker shall appoint a Sergeant-at-Arms.

5.914 The Credentials Committee shall report to the House upon request of the Speaker on:

5.9141 Total number of members of the House eligible to vote.

5.9142 Number of members registered and seated.

5.9143 Announcement of quorum.

5.92 Reference Committees

5.921 The Reference Committees are appointed for a session of House to consider resolutions referred to them.

5.922 The duties of the Reference Committees shall be:

5.9221 To hold open hearings on all referred resolutions at a time and place announced at the first session of the House.

5.9222 To deliberate in closed session on each resolution, and after consideration recommend its final disposition.

5.923 A member of a reference committee who wishes to make a minority report must refrain from signing the majority report and must make his intentions known to the other members of the reference committee while it is in closed session and prior to the presentation of the majority report to the House.

5.93 Other Committees

The Speaker may appoint such other committees as are needed.

6.0 ELECTED OFFICERS

6.1 Designations

The elected officers of the Society are those specified in Article VI of the Constitution. No member shall hold two elected offices in this Society at one time excepting delegates and alternate delegates to the American Medical Association, who may be elected and serve during a concomitant tenure in another elective office of this Society.

6.2 Qualifications

An elected officer must have been for at least two (2) years immediately prior to his election, an active member of the Kansas Medical Society. The Speaker and Vice Speaker of the House shall be elected from among the members of the House.

6.3 Terms

6.31 President Elect

The President Elect shall be elected annually. He shall serve until the next inaugural after his election when he becomes President.

6.32 First Vice President, Second Vice President, Speaker, Vice Speaker, Secretary and Treasurer.

The First Vice President, Second Vice President, Speaker, Vice Speaker, Secretary and Treasurer shall be elected annually, each to serve for one (1) year or until a successor is elected and installed.

6.33 AMA Delegates

Representatives to the House of Delegates of the American Medical Association shall be elected for two-year terms of office which terms shall not expire in the same year. The term of the delegate-elect and alternate delegate-elect shall begin at the annual session of the American Medical Association the year succeeding their election.

6.4 Presidential Succession

If the Presidency is vacated by reason of death, disability for the unexpired term, resignation, or impeachment and conviction, the line of succession shall be the President Elect, the First Vice President, the Second Vice President, the Speaker and the Vice Speaker.

6.41 If the disability of a president is contested, it shall be determined by a two-thirds concurrence of the Council.

6.5 Interim Vacancies

Should vacancies occur in the office of Secretary or Treasurer, the Council shall fill these positions as provided in the By-Laws.

6.6 Nominations

The Council shall annually at the conclusion of the annual session select three (3) physicians who are members of this Society, each representing a different council district other than the districts of the two (2) recent living past presidents to comprise the nominating committee. The Council shall name a chairman. The committee shall meet not later than ninety (90) days prior to the next annual session and present for publication in the *Journal* a list of candidates for each elective office consisting of one or more candidates for the offices of President Elect, First Vice President, Secretary, Treasurer, Speaker of the House of Delegates, Vice Speaker of the House of Delegates, one (1) or more candidates for the office of delegate-elect and alternate delegate-elect to the American Medical Association, and three (3) or more candidates for the office of Second Vice President. The candidates for Treasurer should be named from residents of the vicinity of the central office whenever possible.

6.7 Elections

6.71 Time: The election of officers shall be held at the last meeting of the House of Delegates at each annual session. During the first session nominations for each office are made. When more than two (2) candidates are nominated for any office, a primary election shall be held at the first House of Delegates meeting. The name receiving the fewest votes shall be eliminated and balloting shall continue until not

ad·dic·tion

/ a'dik-shen / 1. Compulsive physiological need for alcohol. 2. In preliminary examinations, could be diagnosed as nervous condition. 3. Arrested through effective alcoholism treatment.

Chances are, during a patient's preliminary examination, alcoholism is not one of your immediate suspicions. But with 10 million Americans suffering from this disease, and over 100,000 in the Kansas City area alone, perhaps it should be. Or perhaps the drinking problem is someone within your patient's family, causing them mental and even physical abuse.

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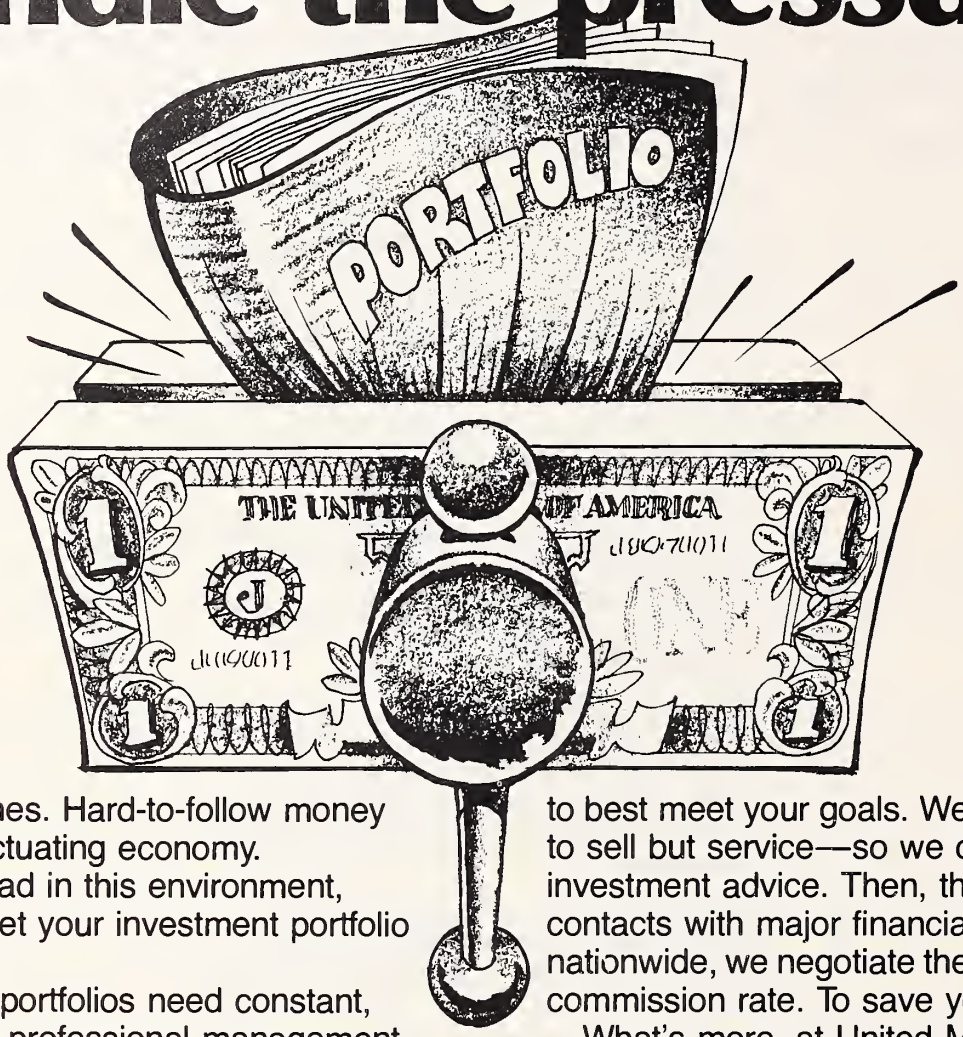
If you have a patient with a drinking problem, send for our detailed informational brochure, or personally contact one of our professional counselors. Inpatient information: Sue Little, Director, Bethany Medical Center, 51 North 12th Street, K.C., KS. 66102. (913) 281-8951. Outpatient: Paul Anderson, Ph.D., Family Institute of Addiction Treatment, 7840 Washington Ave. Suite 300, K.C., KS 66112. (913) 334-6300.

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more than two (2) remain. Tellers shall announce in alphabetical order the names of the two who receive the largest number of votes for each office. In case there exists a tie for second place, another ballot shall be held to select a second candidate. At the last House of Delegates meeting each authorized delegate shall cast his ballot at the time he registers to attend the meeting, and this will constitute the election. In case of a tie, the Speaker, who does not vote otherwise, shall cast the deciding vote.

6.72 Method: All elections of officers shall be by ballot.

6.8 Installation of the Elected Officers

Elected officers, except delegates and alternate delegates to the American Medical Association House of Delegates, shall be installed prior to adjournment of the House at the annual session and shall begin their term of office at the conclusion of the annual session.

6.9 Installation of the President

The oath of office shall be administered to the President Elect during the President's Banquet at the annual session. The President Elect shall become President at the conclusion of the business of the House at the annual session.

6.10 Officials impeached

Officials impeached by the Council are tried by the House of Delegates. A two-thirds vote is necessary for conviction.

7.0 DUTIES OF OFFICERS

7.1 President

The President is the chief executive officer and principal spokesman of the Society. He shall counsel with officers, and others, toward the best interests of the public and this Society, to attempt to further the aims and activities of this Society to the fullest extent. He shall perform such services as custom, necessity and parliamentary procedure require. He shall in accordance with these By-Laws announce his appointments to commissions. He shall be an ex-officio member of all committees and commissions. He shall be extended an opportunity to preside at all major functions of the annual session and shall deliver an address at the time arranged by the Committee on Arrangements. He shall be encouraged to visit the various Council districts during his term of office.

7.2 President Elect

The President Elect shall familiarize himself with the personnel and work of the various committees and of this Society in general. He shall be ready to counsel with the President on matters affecting the future of this Society, and shall otherwise prepare himself for assuming the leadership of this Society at the proper time. Except as is otherwise provided in the By-Laws, he shall appoint a chairman and other members of each commission, and other temporary committees, to serve during his term as President. The complete list of appointments to commissions shall be available for publication and distribution to the membership when he assumes the office of President. The President Elect shall assume the Presidency in the event of the death of the President or the inability of the President to perform the duties of the office. In the event of such succession to office, the President Elect shall not be disqualified from serving his term as President for which he was duly elected.

7.3 First Vice President

The First Vice President shall assist the President in the performance of his duties, shall preside in his absence at the meetings of this Society, or the Council, and shall represent the President in his absence.

7.4 Second Vice President

The Second Vice President shall also assist the President in the performance of his duties.

7.5 Speaker

The House of Delegates shall annually elect from its membership a Speaker and a Vice Speaker whose terms of office begin upon adjournment of the last session of the House and continue through adjournment of the last session at the next annual session. They are eligible for succession.

The Speaker is the presiding officer over all sessions of the House. It is his duty, upon consultation with the Vice Speaker, to appoint reference committees. He shall refer resolutions to reference committees and shall aid delegates and committees to the extent of his ability toward the end that all business of the House may be conducted in an efficient manner.

7.6 Vice Speaker

In the absence of the Speaker or at his pleasure, the Vice Speaker will preside over the House and will perform such duties as would otherwise be performed by the Speaker.

At the first session of the House, nominations shall be placed for the office of Speaker and for the office of Vice Speaker. In the event there are more than two (2) nominations for either office, a primary ballot shall be taken and the names of the two (2) receiving the largest number of votes will appear on the ballot at the last session of the House where the election shall take place.

7.7 Secretary

The Secretary shall advise the Executive Director

in all secretarial matters of this Society and shall act as the corporate secretary for the executive of official documents. He shall perform such duties as are placed upon him by this Constitution and By-Laws, and in the event of death, resignation or removal of the Executive Director, shall assume the duties of that office until the vacancy is filled.

7.8 Treasurer

The Treasurer shall be the custodian of all moneys, securities and valuable papers of this Society. He shall deposit them in safe banking institutions, or invest them, subject to the direction of the Council. He shall be bonded at the expense of this Society in such amount as the House may require. He shall pay all authorized obligations of this Society by vouchers. He may authorize the Executive Director to pay authorized obligations on his behalf. He shall keep a detailed account of all receipts and disbursements, and shall make an annual report to the House concerning the financial transactions of this Society for the preceding fiscal year, the funds of this Society in his care and his actions as Treasurer. He shall subject his accounts to such examinations as this House or the Council may order. He shall establish a revolving fund in an amount approved by the Council for routine expenses of the Executive office, which fund shall be set aside in a separate banking institution and be subject to check by the Executive Director with the understanding that disbursements therefrom shall be satisfactorily accounted by the Executive Director to the Treasurer before replenishment is made.

7.9 Executive Director

The Executive Director shall perform the duties usual to such office except those specifically imposed by this Constitution and By-Laws upon the officers, councilors, committees, commissions, and other representatives of this Society.

He shall be under the employ of the Council, or its representative committee, and in case of his death, resignation or removal, the Council, or its representative committee, shall have the power to fill the vacancy. He shall employ such assistants as the House, the Council, or their representative committees may direct.

The amount of his salary shall be fixed by the Council or its representative committee, with approval of the House of Delegates. He shall be allowed traveling expenses to the extent approved by the Council. He shall use the revolving fund with due regard for efficiency and good business judgment in furtherance of the work entrusted to his care.

He shall be bonded at the expense of this Society in such amount as the Council may require.

He shall:

7.91 Account for, and promptly turn over to the Treasurer all funds of this Society which come into his hands.

7.92 Receive all bills against this Society.

7.93 Investigate their fairness and correctness.

7.94 Prepare vouchers covering the same.

7.95 Forward them, together with proper payment, to the Treasurer, for payment as provided in these By-Laws; or pay them with prior authorization from the Treasurer.

7.96 Keep an account with the component societies of the amount of their assessments, collect the same and promptly turn over the proceeds to the Treasurer.

7.97 Make an annual report of his activities to the House, and shall make such reports as the Council, or its authorized committees may require.

7.98 Within thirty (30) days preceding each annual session, submit his financial books and records to a certified accountant approved by the Council, whose report thereon shall accompany his annual report.

7.99 With the advice of all interested officers, prepare and submit annually to the House a tentative budget of this Society for the ensuing fiscal year, together with the recommendations of the Council, or its authorized finance committee, thereon.

7.910 Subject to instruction by the House, the Council, or the President, he shall act as general administrative officer and business manager of this Society. He shall refer to the proper officials all administrative questions as properly come within their jurisdiction.

7.911 Attend the annual sessions, the meetings of the House, the Council, as many of the committee meetings as possible, and shall keep the minutes of their proceedings.

7.912 Undertake secretarial functions for all officers, councilors, committees and commissions of this Society, and shall assist wherever possible in the performance of their duties.

7.913 With the cooperation of the secretaries of the component societies, he shall keep a record of all legally licensed doctors of medicine in this state, together with such information as is available about each. He shall transmit to the American Medical Association all copies of records that may be desired by that association, together with such other information as may be of value.

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Getting Ready for the Office of the Future: Factors to Consider Now.

The ultimate "Office of the Future" may still be years away. But the information systems you now have in place – or add in the short term – will have a big effect on how quickly your office advances into the future.

Let's examine some factors to consider as new technology unfolds.

Business Solutions

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Most simply, the office of the future is a technological solution to business problems. And that's where your application of information systems should begin.

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What is State-of-the-art?

There is an important difference between functional state-of-the-art and technological state-of-the-art. Some systems which are technologically advanced do not offer any significant advantage in their performance. When comparing systems, the technology used is not as important as what the system can do for you. And, since the costs of different technologies vary, you may be paying a premium for something that is no more functional than systems utilizing less expensive technologies. On-premise digital switching is one of them.

The Digital Age

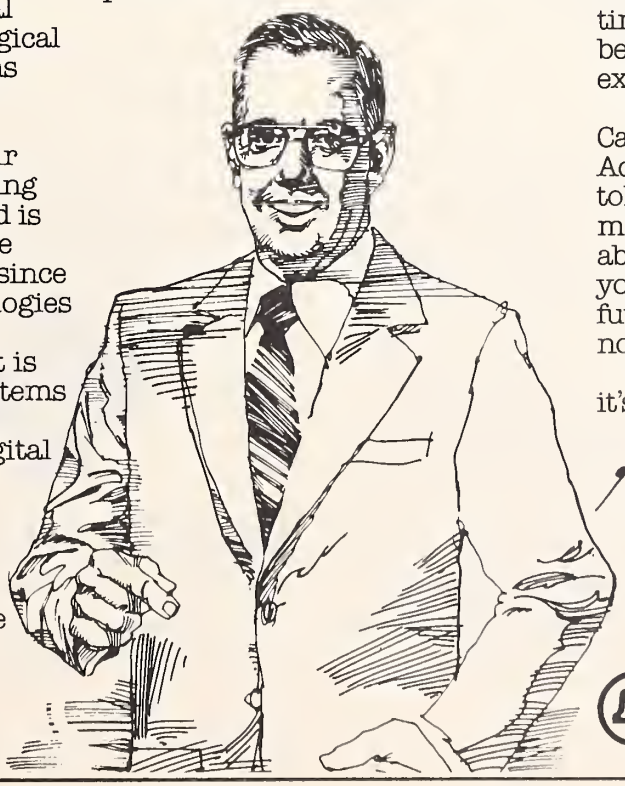
There's no doubt that in the future both voice and data communications will be transmitted in digital form. But, it's not a digital world yet. In fact, 85 percent of what goes on in the office today is analog (voice). And this will not change dramatically in the near future. So – today and in the near future – a digital switching system will rarely improve transmission quality and speed.

But Bell is already leading the way into a digital world. About 40 percent of our metro exchange facilities are digital. Sixty percent will be by the mid-eighties. And, Bell's Dataphone® Digital Service now serves 350 cities encompassing 95 percent of the nation's data center installations.

Systems that Keep Pace

In preparing for the office of the future, it's important that your systems' software can be updated to take advantage of new technology. This will reduce the number and cost of product hardware change-outs. Bell's focus is to enhance functions by offering you more and better software applications.

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Integration, Compatibility

"Office of the Future" implies the integration of internal office systems, and external intercity or even international networks. It also implies the integration of all office information management such as voice, data, video, text and sensor. All of this will be driven through a central controller system.

Because of this integration, a major consideration in planning an information network is compatibility. Currently, there is no industry standard for common interfaces between technology or even between digital systems. And the bit rate necessary to transmit digital voice messages is decreasing due to constantly evolving improvements.

Integrated Planning

It's important for businesses to have integrated planning that encompasses the entire area of information management rather than just telecommunications or data processing. Information, like other business assets, must be properly organized, controlled and distributed to make it a productive management tool.

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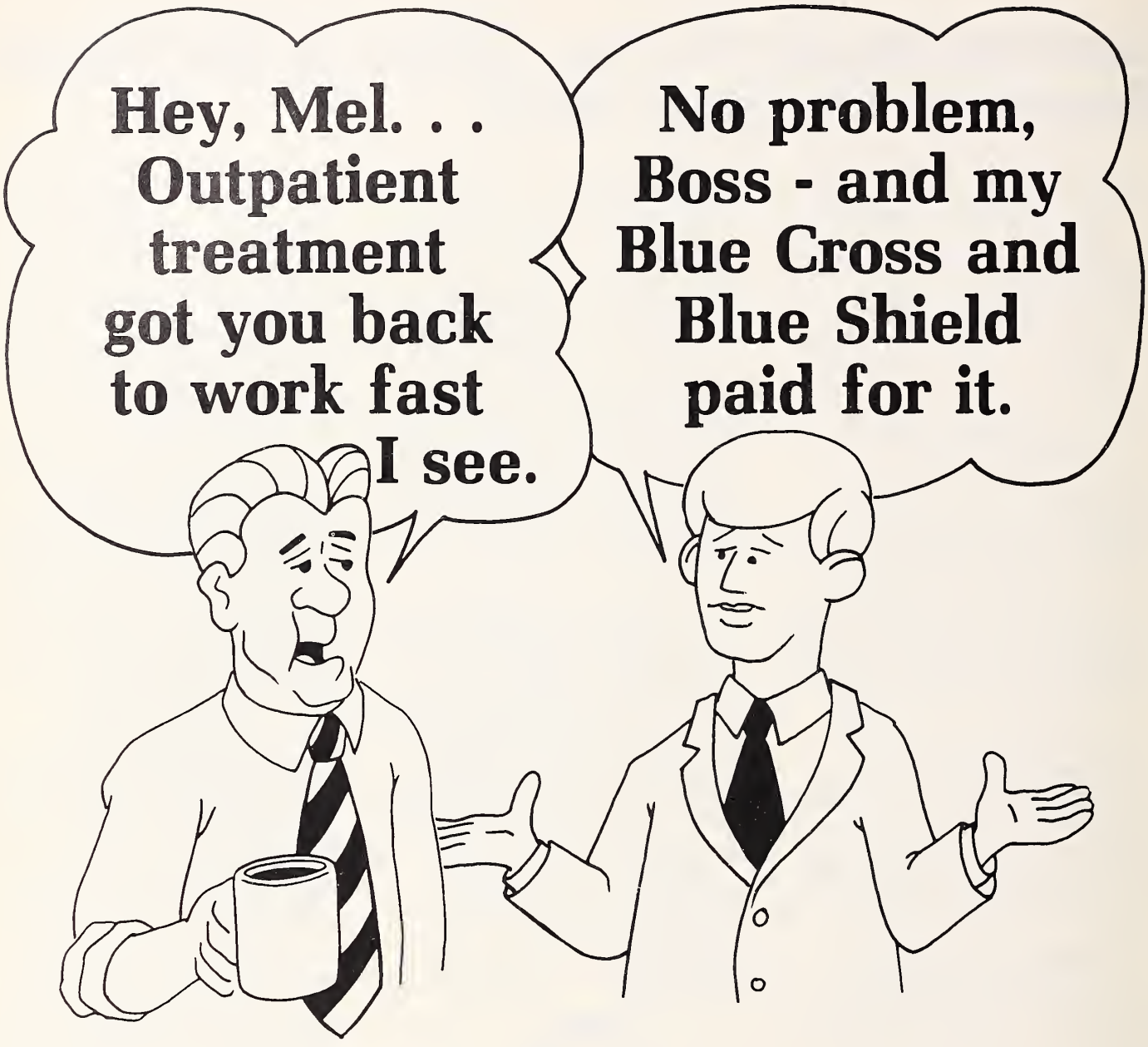
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7.914 Endeavor to visit component societies when his duties will permit, or when an emergency requires personal attention, and shall keep the officers of the component societies informed about the activities of this Society and of the medical profession in general by the issuance of bulletins.

7.915 Supply the component societies with necessary forms and blanks for conducting their official business with this Society.

7.916 Inform the Society upon all pending or enacted legislation and upon activities of governmental offices and agencies affecting the medical profession and public health.

7.917 Secure, upon invitation, medical speakers to address lay organizations on subjects which are in accord with the aims and ideals of this Society. When requested, he shall assist the component societies in securing speakers and in preparing programs.

7.918 Notify all members of meetings, officers of their election, and committees of their appointment and duties.

7.919 Upon authorization by the Committee on Arrangements, he shall prepare and issue an official program for each annual session.

7.920 Provide for the registration of members and delegates at each annual session.

7.921 Be the custodian of the general papers and records of this Society except as properly belong in the custody of other officials.

7.922 Conduct the official correspondence of this Society, and shall sign all authorized communications.

7.923 Aid the councilors in organizing and improving the component and district societies, and in the extension of the usefulness and influence of this Society.

7.924 Act as business manager of the *Journal of the Kansas Medical Society* under supervision of the Editorial Board, and in a similar capacity to the extent authorized for other publications of this Society.

7.925 Perform any additional duties as may be required by the House, the Council, their committees or the President.

8.0 THE COUNCIL

8.1 Composition

8.11 Members of the Council are the President, President Elect, First Vice President, Second Vice President, Secretary, Treasurer, and Speaker and Vice Speaker of the House, Delegates and Alternate Delegates to the American Medical Association, and a councilor from each Council District. Each councilor and an alternate are to be elected by members of the component societies of the district prior to the convening of the House of Delegates; excepted, that the councilor or his alternate may be elected by a caucus of the delegates if the members have not done so by the date the House of Delegates convenes. The elected alternate replaces an active member in his absence.

8.12 Associate membership of the Council includes alternate councilors and one (1) representative each from the University of Kansas School of Medicine, the Kansas State Board of Health, the Kansas State Board of Healing Arts, the President of Kansas Blue Shield, and one (1) representative each from recognized specialty organizations. Associate members may attend plenary sessions of the Council but shall not be entitled to vote.

8.13 The Executive Director of the Society attends all sessions of the Council as an administrative advisor.

8.14 Advisory members of the Council are the Editor of the *Journal,* chairmen of the Society commissions, and past presidents. They may attend plenary sessions of the Council, but are not entitled to vote.

8.15 The Executive Committee of the Council shall be composed of the President, the President Elect, the Immediate Past President, the First Vice President, the Second Vice President, the Secretary, the Treasurer, the Delegates to the AMA, the Speaker and Vice Speaker of the House of Delegates. The President of the Kansas Foundation for Medical Care shall be an ex-officio, non-voting member. The committee shall meet regularly and at least six (6) times during each year at the call of the President, and shall have authority to act in the interim between meetings of the Council upon all matters which would ordinarily require approval by the Council, which do not properly necessitate a special meeting of the Council and which have not been delegated elsewhere by these By-Laws.

8.2 Meetings

8.21 The Council meets at the conclusion of each annual session of the House of Delegates and at the call of the President during the year. A meeting is convened upon petition of five (5) active members of the Council.

8.22 Business may be transacted when a quorum is present. A majority of the active members is a quorum.

8.23 Adequate notice must be given the members by the Executive Director of the time and place of the meeting.

8.3 Duties

8.31 Between annual or special sessions of the House of Delegates, the Council or the Executive Committee of the Council will transact business on behalf of the Society. Council or Executive Committee action may not conflict with former action of the House of Delegates. Transactions of the Council otherwise have the same authority as those of the House of Delegates except they cannot bind the Society beyond its next annual session.

8.32 The Council is authorized to issue or revoke charters to component societies in the name of the Society. The council for the district of origin presents the application for charter to the Council. The application must contain the following information:

8.321 The geographical area to be included in the proposed component society.

8.322 Certification by the presiding officer of each component society within the area of the new society that his members have voted to relinquish the charter in effect.

8.323 A guarantee that all members affected will retain initial membership in the new society, but be subject to its by-laws subsequently.

8.324 The name of the new society; all to be attested by the signatures of the elected officers.

8.33 The Council shall supervise the publication of the *Journal of the Kansas Medical Society*. It appoints an editorial board of five (5) members, one of whom is named Editor (See 10.0).

8.34 The Council is responsible for the funds and expenditures of the Society. The Treasurer submits to it a budget of estimated expenses for the coming year. Appropriation of money for special purposes not previously budgeted must be authorized by the Council.

8.35 The Council sets the amount of dues for the coming year, directing the Executive Director to inform component societies no later than October 31.

8.36 The Council may impeach elected officers and councilors.

8.37 The Council is responsible for the determination, five (5) years in advance, of the time and place of the annual session of the Society.

8.38 The Council employs and supervises the activities of the Executive Director.

8.39 The President of the Society presides over the Council. In his absence the First Vice President will preside.

8.310 The Council may create committees from its membership to act between Council meetings. Special committees may include additional members of the Society who are not Council members. When the Society is invited to send a representative, or committee for service outside its own jurisdiction, the appointment(s) is made by the Council except in emergency when the Executive Committee may appoint.

8.311 All money of the Society is paid to the Treasurer who is accountable for it. The Council may inspect or audit the accounts of the Treasurer at any time, but at least annually; it may review or audit the expenditure of any officer, board or committee, and insure annual reports to the House of Delegates on all expenditures of the Society.

8.312 If the office of Secretary or Treasurer is vacated, the Council appoints a successor for the unexpired term. To protect the new Treasurer, an audit is conducted at once. If a councilor position is vacated, the corresponding alternate sits as councilor until the annual session. Then a councilor is elected as provided above to complete the three (3) year term. The alternate so seated becomes a voting member of the Council. If the incomplete term is for less than two (2) years, the elected councilor is eligible for two (2) more terms.

8.313 A summary of Council activities and reports to it between sessions of the House of Delegates is submitted to the House at each annual meeting.

8.4 Council Districts

Council Districts are comprised as follows:

8.41 *District #1:* Atchison, Brown, Doniphan, Jackson, Jefferson, Leavenworth, Marshall and Nemaha Counties.

8.42 *District #2:* Wyandotte County.

8.43 *District #3:* Johnson County.

8.44 *District #4:* Allen, Bourbon, Cherokee, Crawford, Labette, Montgomery, Neosho, Wilson and Woodson Counties.

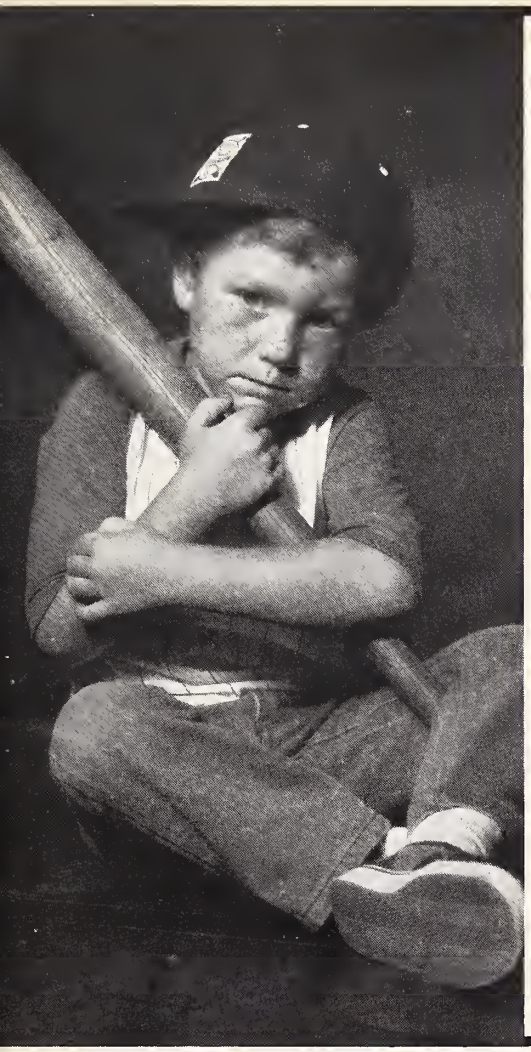
8.45 *District #5:* Clay, Geary, Pottawatomie, Riley and Washington Counties.

8.46 *District #6:* Shawnee County.

8.47 *District #7:* Chase, Coffey, Lyon, Morris, Osage and Wabaunsee Counties.

8.48 *District #8:* Butler, Cowley and Greenwood Counties.

8.49 *District #9:* Cloud, Dickinson, Jewell,



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Mitchell, Ottawa, Republic and Saline Counties.

8.410 *District #10:* Harvey, McPherson, Marion, Reno and Rice Counties.

8.411 *District #11:* Sedgwick County.

8.412 *District #12:* Barber, Harper, Kingman, Pratt and Sumner Counties.

8.413 *District #13:* Ellis, Ellsworth, Graham, Lincoln, Ness, Osborne, Phillips, Rooks, Rush, Russell, Smith and Trego Counties.

8.414 *District #14:* Barton, Edwards, Pawnee and Stafford Counties.

8.415 *District #15:* Clark, Comanche, Ford, Gray, Hodgeman, Kiowa, Meade and Seward Counties.

8.416 *District #16:* Cheyenne, Decatur, Gove, Logan, Norton, Rawlins, Sheridan, Sherman, Thomas and Wallace Counties.

8.417 *District #17:* Finney, Grant, Greeley, Hamilton, Haskell, Kearney, Lane, Morton, Scott, Stanton, Stevens and Wichita Counties.

8.418 *District #18:* Anderson, Douglas, Franklin, Linn and Miami Counties.

8.419 *District #19:* Allen, Chautauqua, Elk, Montgomery, Neosho, Wilson and Woodson Counties.

8.5 Notification of Councilor Election

The Executive Director will notify all component societies in a given Council district three (3) months prior to the annual session at which its councilor term ends. A poll may be held in the district before the annual session to select the new councilor whose official election is held as provided above.

8.6 Councilor Term of Office

Each councilor is elected for three (3) years. He may serve two (2) successive terms except as limited above (8.313). On one year, terms begin for councilors in Districts 1, 3, 5, 8, 9 and 17; the next year in Districts 2, 4, 11, 13, 14, 15 and 19; the third year in Districts 6, 7, 10, 12, 16 and 18.

8.7 Duties of the Councilor

The councilor represents the members of his district in the government of the Society. He is expected to know their needs and represent them fairly. Conversely, he represents the Society to the members of his district, giving rise to some of these duties by which he is bound but not limited:

8.71 He must afford societies within his district complete understanding of Society policy and activities.

8.72 He must visit each component society in his district at least once annually.

8.73 He must function as censor and peacemaker in his district.

8.74 He must aid in improving the organization of the societies in his district for their own efficiency and to augment the proper function of this Society.

8.75 He will call a district meeting at the request of the President.

8.76 He is expected to attend meetings of the Council. If he cannot be present, he must notify his alternate who will sit in the Council for him and exercise the vote of his district. His failure to notify his alternate after two (2) consecutive absences will represent resignation and the component societies in the district will select his successor to fill the unexpired term of his appointment.

8.77 He is obliged to teach his alternate the duties of councilor, to acquaint the alternate with Council policies and procedures, and to keep him informed of current Society transactions.

8.78 He serves as a voting member of the House of Delegates during his Council tenure (See 5.24).

9.0 COMMITTEES AND COMMISSIONS

Commissions and Committees are appointed by the President for a one-year term. The Chairman, size and membership of such commissions and committees shall be designated by the President. Commissions and Committees may introduce resolutions for consideration by the House of Delegates.

9.1 Judicial Committee

There shall be a Judicial Committee consisting of seven (7) members, representing different specialties, serving terms of three (3) years in length with a limit of two consecutive full terms. One member of the committee shall be a physician representing the Kansas State Board of Healing Arts. The President shall appoint the committee, from names submitted by the specialty societies, and such appointments are subject to approval by the Council. The terms shall be staggered to assure continuity. The majority of the Judicial Committee shall constitute a quorum and the affirmative vote of a majority of those members present shall constitute the action of the Judicial Committee.

9.11 The Committee shall invite as a temporary, ex-officio member the councilor from whose district a complaint or possible cause of action shall have been submitted. In the event that the councilor is the subject of the complaint or cause of action, the alternate councilor shall sit in his place.

9.12 In the event that any member of the Committee is the subject of the complaint or cause of

action, he shall be excluded during the Committee's deliberations and action on such complaint and from Committee membership, but shall otherwise retain the same rights and privileges as any other member of this Society.

9.13 The Judicial Committee is charged with the responsibility of reviewing and acting on grievances or matters of ethics involving a member of this Society. The committee will conduct such inquiries and investigations as are needed to render timely decisions on matters referred to it. Decisions of the committee may be appealed to the Council. The decision of the Council on such appeals is final and subject to no higher appeal within this Society. The committee may utilize such consultants and advisory committees as it may require.

9.14 The Judicial Committee shall receive written complaints from any person, whether or not he or she is a physician, a member of this Society, a patient of a physician, or any other person, lay or professional.

9.15 After investigation of, impartial hearing of, and deliberation upon a complaint, the Judicial Committee by majority vote, may:

9.151 Consider the case closed.

9.152 Recommend settlement.

9.153 Express its advice to a member of this Society on any matter pertaining to his or her professional conduct.

9.154 Recommend to the Council of the Society:

9.1541 That it take action to expel, suspend or reprimand a member.

9.1542 Submit proposed charges to an appropriate state or federal law enforcement agency.

9.1543 Submit the case and findings to the Kansas State Board of Healing Arts.

9.16 Upon reaching a decision, the Judicial Committee chairman shall transmit to the Council a written statement and explanation of the final decision as soon as possible after the committee has completed the investigation of the case and has arrived at its decision.

9.17 The Judicial Committee shall keep a complete record of all complaints, answers, findings and final disposition of its decisions and disposal of complaints, and shall prepare an annual report to the Kansas Medical Society.

9.18 The Judicial Committee shall not at any time exceed or take by assumption any authority other than that granted by this Constitution and By-Laws.

9.2 Committee on State Meeting Format

The Committee on State Meeting Format is comprised of the most recent general chairman of the annual session from each city where sessions are held. The term of office ends when a new general chairman is appointed by the respective component society. The President Elect is chairman of this committee which meets not later than sixty (60) days after each annual session in the host city for the next session. The meeting is held to review with the local planning committee plans for the coming session. The committee further recommends a general meeting plan to the host society selected for two (2) years later.

9.3 Policy Statements

Public statements of policy by commissions or committees have only the force of recommendations, becoming Society policy after approval of the House of Delegates or the Council.

9.4 Expenditures

Expenditures by commissions or committees must have prior approval of the House of Delegates or the Council to be binding on the Society. No contract or monetary obligation shall be incurred in the name of the Kansas Medical Society by any member, employee or agent until he is authorized by the House of Delegates, the Council or the Executive Committee. Authorization cannot extend beyond the next annual session.

10.0 THE EDITORIAL BOARD

10.1 Composition and Appointment

The Editorial Board is comprised of five (5) or more members, appointed for terms of three (3) years by the Council; no more than two (2) appointments for a complete term are made in a single year. When a vacancy occurs it is to be filled for the unexpired term at the next meeting of the Council.

10.2 Duties of the Chairman

10.21 To direct the operation of the Board.

10.22 To be the Editor of the *Journal of the Kansas Medical Society*.

10.23 To make an annual report to the House of the activities of the Board and the status of the *Journal*.

10.3 Duties of the Board

10.31 To edit, compile, publish and distribute all publications of this Society and make business arrangements for publication as authorized by the House of Delegates or the Council.

10.32 The Board may appoint associate editors for the *Journal* from any or all Council districts.

Associate editors will assist the Board in securing information for publications, in forwarding suggestions for improvement of the *Journal,* and supplementing the business functions associated with publications.

10.33 The Board with its associate editors will meet each year at the annual session and at the call of the chairman.

10.34 The Board collects and records data on deaths of Society members and other non-member physicians in the state. Data is published as it is received and a compilation of the previous year's deaths is reported at the annual session.

10.4 Financial Regulations

10.41 The Board is enjoined to maintain the *Journal* as nearly self-supporting as possible, but as the official publication of the Society, it deserves the financial assistance necessary to correct a reasonable deficit. If a deficit exceeds budgeted money, a financial report is sent to the Council which may appropriate additional funds.

10.42 Accounting procedures of the *Journal* and other publications will be kept in separate ledgers and its accounts shall be maintained apart from other Society funds.

10.43 Expenditures authorized by the Board are paid by vouchers signed by the chairman and countersigned by the managing editor or the business manager of the *Journal*. The three signatories will be individually bonded for a sum not less than five thousand dollars ($5,000). Certification of vouchers is submitted to the treasurer of the Society monthly.

10.44 Surplus funds accruing to the *Journal* or other publications at the end of the fiscal year are deposited to a reserve account within limits established by the House of Delegates or the Council. Surplus funds beyond that limit will revert to the general fund of the Society.

11.0 COMPONENT SOCIETIES

11.1 Definition

Component societies are groups of physicians organized in the State of Kansas, adopting principles of organization in accordance with these By-Laws and receiving a charter from this Society.

11.2 Charter Requirements

11.21 Each county shall be included among the component societies in this state, but only one (1) component society may be chartered in a county.

11.22 New component societies will not be chartered with fewer than ten (10) members.

11.23 Physicians in counties with too few physicians for a successful organization may affiliate with those of adjoining counties to form a multicounty component society.

11.24 Charters are granted to component societies upon action of the Council as provided above (8.32).

11.25 Following Council recommendation, the House of Delegates may revoke the charter of a component society whose actions conflict with these By-Laws.

11.26 If the active membership of a component society falls below four (4) in number, its charter shall be revoked and the members reassigned by the Council to one of the following alternatives:

11.261 Attach the entire membership to the nearest component society.

11.262 Attach the entire membership to any adjacent medical society by unanimous request of the members.

11.263 Attach the members separately to adjacent medical societies for reasonable cause.

11.3 General Regulations

Component societies may set rules for membership qualification, organization and transaction of business except as specifically limited in these By-Laws.

11.4 Regulations For Membership

11.41 Since membership in this Society is dependent on that of the component society, any reputable and ethical physician with the degree of Doctor of Medicine or Osteopathy, or their equivalent, from an accredited medical or osteopathic school, fully licensed to practice medicine and surgery by the Kansas State Board of Healing Arts, and the majority of his professional work being conducted in the state, shall have the privilege of applying for component society membership. Also eligible for membership shall be students attending accredited medical schools in Kansas.

11.42 A physician may not hold active membership in two component societies.

11.43 No physician may be an active member of a component society without becoming a member of the Kansas Medical Society.

11.44 A physician living in one county but conducting his practice in another is eligible for membership in the county where the majority of his work is performed. He may maintain active membership for reason of convenience in the county of his residence only with the permission of the society within whose jurisdiction he practices.

11.5 Movement Within the State

A member of any component society, upon establishing the majority of his practice in the area of another component society, may retain his original membership only with the permission of the society into whose jurisdiction he moves.

11.6 Transfer of Membership

A member desiring to transfer membership from one component society to another applies to the new society in the same manner as an original applicant. His application is accompanied with an appropriate recommendation and a report of his standing in the previous society, attested by the president. When the application has been acted upon, the secretary will notify the applicant at once of the society's decision. The secretary of the previous society is also notified since acceptance by the new society requires that he be dropped from the roll of previous membership. Rejection by the new society does not affect membership status in the previous society.

11.7 The Annual Report

The secretary of each component society will maintain a roster of its membership and of non-affiliated registered doctors of medicine or osteopathy within its boundaries. The roster must include for each member his address, medical or osteopathic school and date of graduation, date of license to practice in Kansas, and the dates of past changes in membership status. Only the names and addresses of non-members need be recorded.

On or before the first day of February, the secretary shall forward a current roster to the Executive Office, including notations of death or change of membership status among members and, as far as possible, among non-members.

11.8 Dues and Assessments

11.81 All dues owed the Society should be remitted before the first of February of each year unless permission for a delay is secured from the President. The remittance may not be delayed beyond the first of April of the same year. The number of delegates of all component societies shall be determined on the basis of information regarding membership available to the state society office as of March 15 of each year; and a list of delegates and alternates shall be submitted to the Executive Director no later than March 20 of each year for the purpose of publishing the names in the program of the annual meeting in May. Assessments are due under the same rule unless special provisions are set by the Council.

11.811 A member delinquent in the payment of his dues on April 1 of that dues-paying year shall be suspended.

11.82 Individual checks of funds for dues to the American Medical Association are transmitted from the treasurer of the component society through the Executive Office to the proper destination.

11.83 A component society failing to pay its dues or assessments according to these By-Laws is suspended from membership in the Society until the requirements are met or until written certification of the suspension of members in arrears is received by the Executive Office. A member remaining in arrears beyond December 31 of the year in which he became delinquent shall be expelled.

11.831 Any member dropped from the membership rolls for delinquent dues shall be readmitted only after applying as a new member; provided that the Executive Committee shall be empowered to consider exceptional cases on an individual basis whether a member must pay dues for the years in which he/she became delinquent should he/she desire to renew membership in the future.

11.84 A component society suspended from membership in violation of this section is not eligible collectively or individually to participate in the proceedings of this Society nor seats in the House of Delegates until the deficiency has been corrected.

11.85 Punitive action will not be instituted against a component society certifying that members in arrears have sufficient cause from illness, leave of absence, or otherwise, which prevents the practice of their profession. If the component society finds cause to waive the local dues of a delinquent member, a written account should be sent to the Executive Office. The Council will then determine whether the waiver should be properly extended to the dues of the Society. The secretary of the concerned component society will be notified of the Council decision by its district councilor.

12.0 RULES OF ORDER

Deliberations of this Society and its subsidiary Council, sections, commissions and committees shall be governed by these By-Laws, and when not otherwise specified by the provisions of Sturges' Standard Code of Parliamentary Procedure.

"The Rules of Order and By-Laws of this Society may be suspended at any time by a vote of two-thirds of those delegates present."

13.0 OFFICIAL STATEMENTS

Memorials, resolutions or opinions of any character which conflict with policies of the House of Delegates shall not be issued in the name of the Kansas Medical Society. No obligation, oral or writ-

ten, shall be incurred in the name of the Kansas Medical Society by any member, employee or agent unless the same has been previously authorized by vote of the House of Delegates, or the Council, or the Executive Committee and no such authority shall be extended beyond the next annual meeting of the House of Delegates.

14.0 MEDICAL ETHICS

The Principles of Medical Ethics of the American Medical Association shall govern the conduct of members.

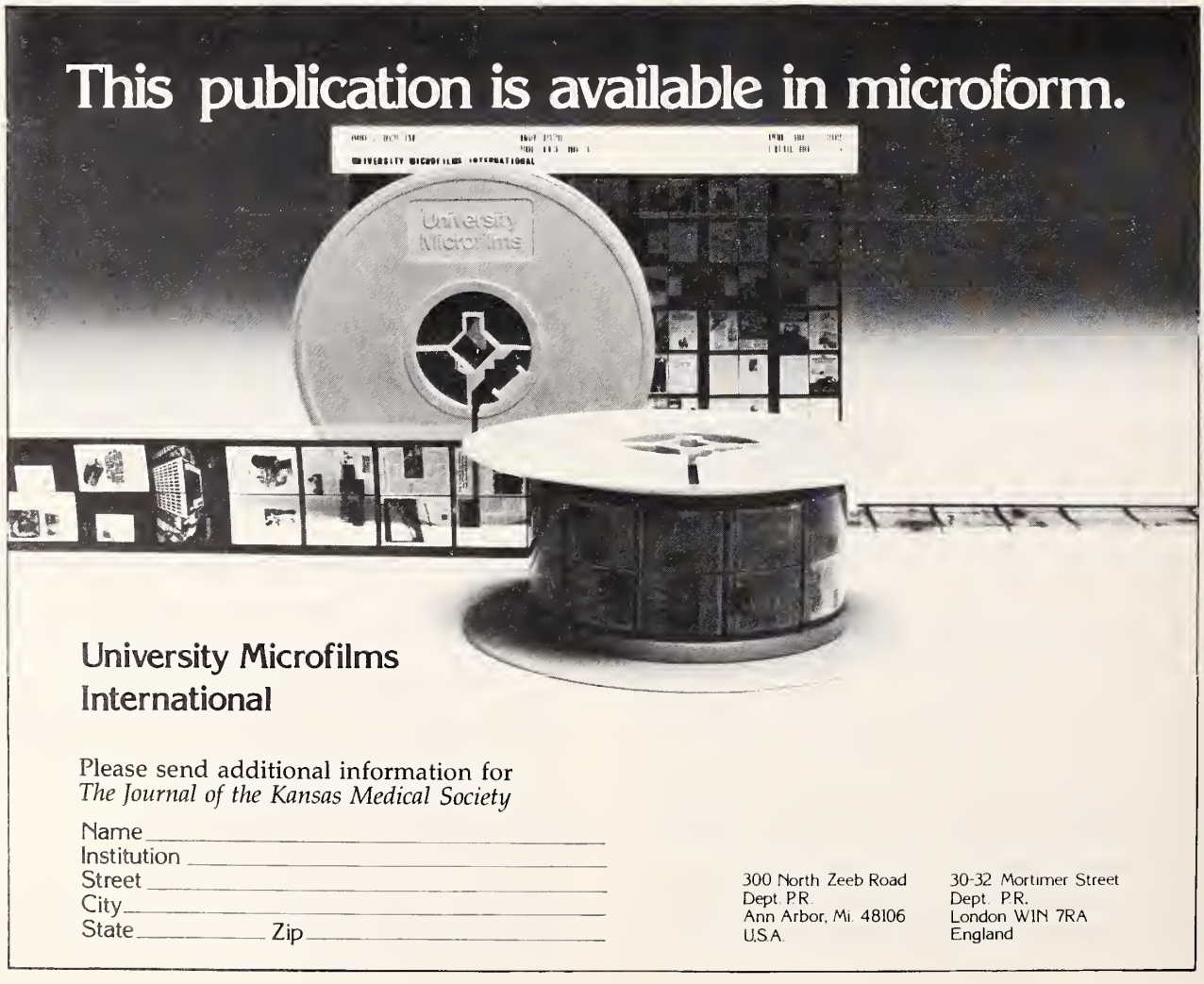
15.0 AMENDMENTS

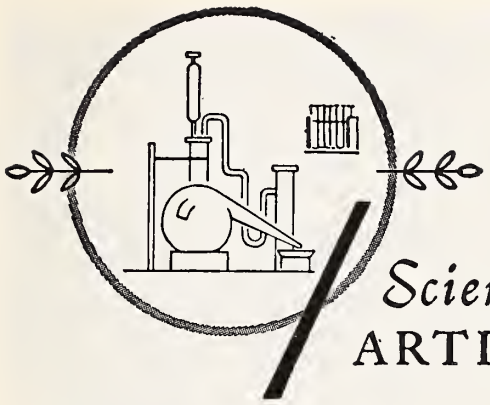
These By-Laws may be amended by a two-thirds majority of the House of Delegates present provided such amendment has been considered by a reference committee.

RESOLUTION OF ADOPTION

Be it hereby resolved that this revised Constitution and By-Laws shall be in full force and effect at the close of the meeting of its adoption and shall supersede all prior Constitutions, By-Laws and Amendments.

Likewise, officers, councilors, board and committee members holding offices for definite terms under the previous Constitution and By-Laws shall serve until expiration of term for which elected and until their successors, as provided by this revised Constitution and By-Laws, are duly elected, qualified and installed.





Percutaneous Transluminal Angioplasty

The Treatment of Stenotic or Occluded Iliac Arteries

JOSEPH P. GALICHIA, M.D.; GREGORY F. DUICK, M.D.; ASHOK K. BAJAJ, M.D. *and* **ROGER W. ROBERTS, D.O.,** *Wichita*

Percutaneous transluminal angioplasty (PTA) was performed in 47 patients with a total of 66 iliac artery lesions. There were 63 common or external iliac artery stenoses and three totally occluded common iliac arteries. In 57 instances, the treatment was successful; there were three major complications. PTA is a cost effective procedure, especially suited for short segment iliac stenotic lesions.

PERCUTANEOUS transluminal angioplasty (PTA) is being increasingly utilized in this country. In 1964, Dotter and Judkins first established modification of atherosclerotic plaques by passing progressively larger catheters across a narrowed arterial lesion transluminally.[1] Since the introduction of the balloon catheter by Grüntzig and co-workers in 1976, PTA has gained wide acceptance.[2] Using these newer Grüntzig catheters, PTA has been used in treating lesions in the coronary, iliac, femoral, renal, axillary, superior mesenteric, and the subclavian arteries.[3]

During the period from February 1980 to March 1982, we used the Grüntzig method for dilatation of 66 highly stenotic or totally occluded common or external iliac arteries in a total of 47 patients. This report reviews our early experience with this non-surgical mode of treatment. PTA was attempted as treatment for atherosclerotic narrowing in these patients with disabling claudication or rest pain. All patients had indications for aortofemoral bypass grafting, but many were poor surgical candidates because of other concurrent medical illnesses. Prior to PTA, non-invasive studies, aorto-femoral angiograms, and surgical consultations were obtained. Our patients included 33 men and 14 women, ranging in age from 40 to 84 years of age *(Table I)*. Initial successful dilatation was defined as improvement of the arterial lesion by angiography, post-PTA, and improvement of clinical symptoms during the hospital course.

TABLE I
PATIENT RISK FACTOR DISTRIBUTION

	Number	*Mean Age*	*Smokers*	*Hyper-tension*	*Diabetes Mellitus*
Male	33	59.3	29 (87.9%)	17 (51.5%)	4 (12.1%)
Female	14	62.6	8 (57.1%)	8 (57.1%)	8 (57.1%)
Total	47	60.3	37 (78.7%)	25 (53.2%)	12 (25.5%)

From the Department of Cardiology, St. Francis Hospital, The University of Kansas School of Medicine-Wichita.

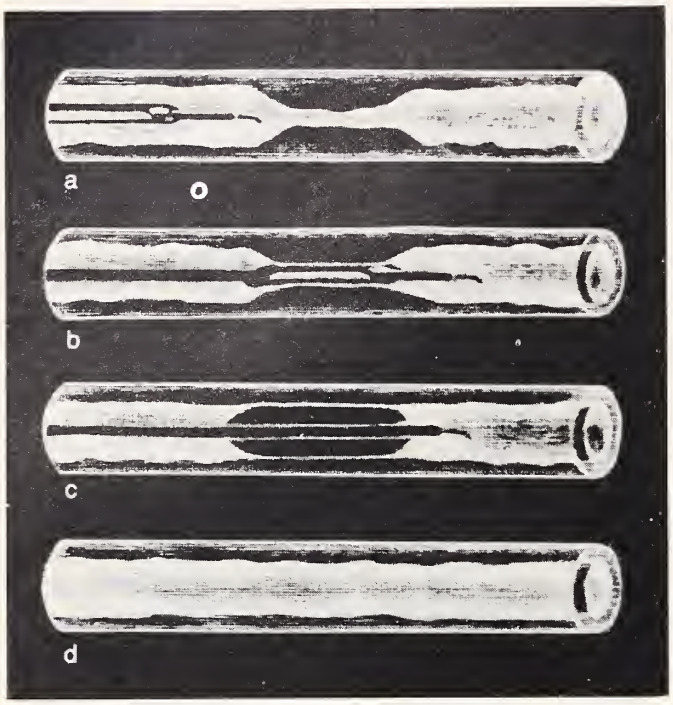


Figure 1. The guide wire with the angiographic catheter is introduced into the vessel (a). The lesion is carefully crossed and the angiographic catheter replaced by a Grüntzig catheter (b). The balloon is repeatedly inflated at lesion (c). After PTA, the luminal diameter is seen to increase (d).

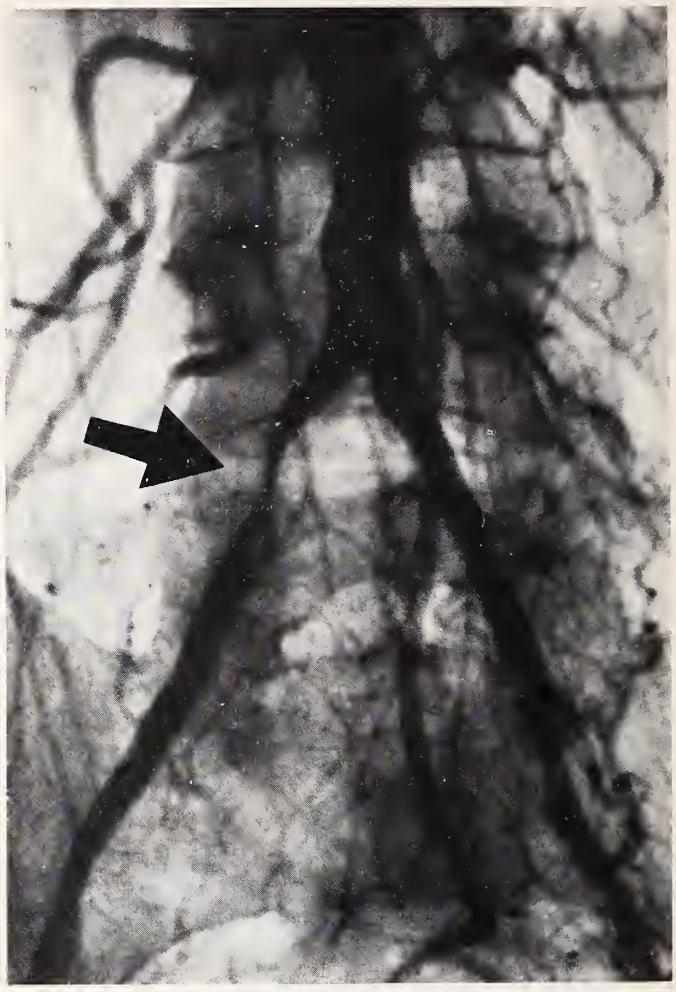


Figure 2. Before PTA, there is a 90 per cent segmental stenosis of the right common iliac artery.

Methodology

A satisfactory angiogram of the aorta, common iliac, and the femoral vessels was obtained for each patient. At the time of angiography, or at a later date, the affected vessel was dilated utilizing a Grüntzig catheter. The ipsilateral femoral artery is entered percutaneously with a thin walled Cook needle. A J-tipped guide wire is introduced into the vessel, and a #6 straight angiographic catheter is introduced over the wire *(Figure 1)*. After obtaining the distal pressure, the J guide wire with the catheter is carefully advanced across the lesion. The proximal arterial pressure is obtained and the angiographic catheter is replaced, leaving the guide wire proximal to the lesion, with an appropriate Grüntzig catheter. The balloon segment of the catheter is placed across the stenotic lesion. The balloon is repeatedly inflated, using a USCI calibrated inflation pump, beginning at 3 and then progressing to 4-5 atmospheres of pressure. Each inflation is sustained for 40-60 seconds. Changes in the caliber of the vessel can be seen angiographically during inflation and facilitates immediate evaluation of PTA. A control angiogram using the same catheter and pull-back pressures are monitored to evaluate the results of PTA. Totally occluded segments are transversed with the guide wire in the J configuration, when possible. Following PTA, the puncture site is manually compressed for 10-15 minutes. The patient is kept at bedrest for six hours, and intravenous heparin is administered for 12-48 hours to prevent thrombosis. Aspirin and dipyridamole (Persantine) are administered following the procedure and for a six month period afterwards. In the early patients, warfarin (Coumadin) was used for anticoagulation after discharge.

A 65-year-old white male presented with right hip pain existing for five years, which had progressed to pain upon walking 40-50 yards. He had a 40-pack-year history of smoking and a ten year history of treated hypertension. He also had a medical history of right carotid endarterectomy, angina, congestive heart failure, chronic obstructive pulmonary disease, and obesity. Physical examination revealed the

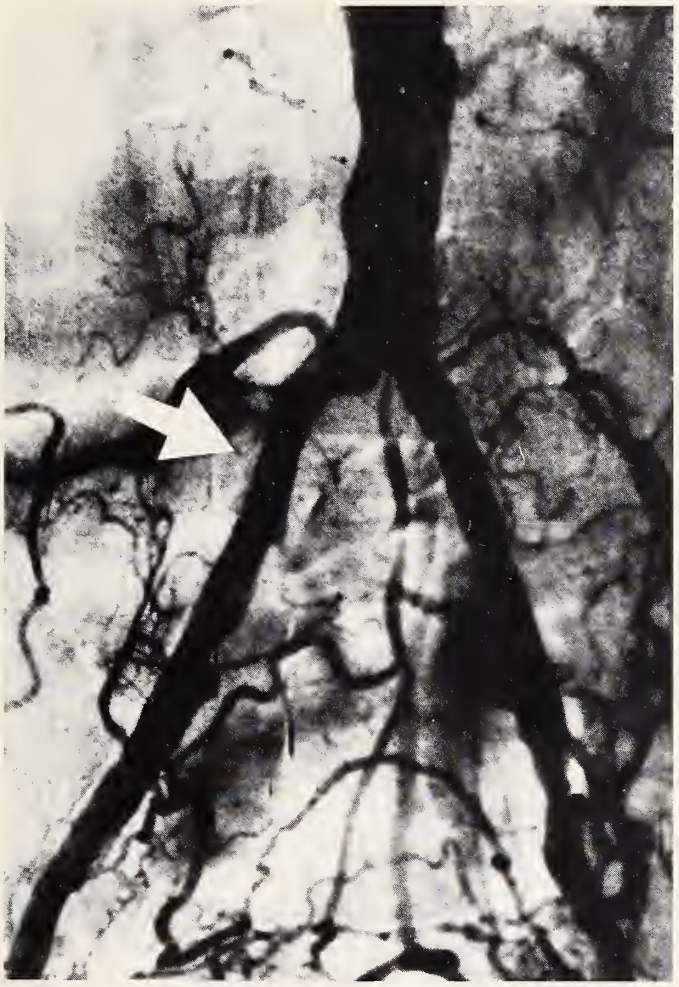


Figure 3. After PTA, the lumen of the right common iliac artery is markedly improved.

right femoral pulse to be markedly decreased. The right popliteal, posterior tibial, and dorsalis pedis artery pulses were totally absent. A non-invasive study showed right sided claudication upon exercise and was compatible with a hemodynamically significant proximal right arterial lesion. An aortogram revealed 90 per cent short segment stenosis of the right common iliac artery *(Figure 2)*. The patient was treated by percutaneous transluminal angioplasty with marked improvement of the luminal diameter *(Figure 3)*. There was total resolution of the patient's symptoms.

Results

As shown in *Table II*, we were able to dilate 55 (87.3%) of the 63 highly stenotic iliac lesions with good angiographic and hemodynamic improvement. Two of the three occluded common iliac arteries were opened and dilated successfully. We were unable to cross four lesions — three highly stenotic vessels and one totally occluded — due to vessel tortuosity or extreme stenosis, and the procedure was terminated without complications. In one patient there was poor distal run-off, post satisfactory dilatation, in the lesion itself. In one patient, a vasovagal reaction occurred and persisted despite anticholinergics, and the dilatation procedure was postponed. The patient was brought back three days later and the lesion was satisfactorily dilated. In another patient, during angioplasty, several radiopaque fragments were seen to embolize distally. The femoral catheter was left in place and the patient was treated with overnight streptokinase infusion. The next morning, the patient's symptoms had totally resolved.

Two patients had unsuccessful attempts due to serious complications, and underwent aorto-femoral bypass grafting. The first of these patients was a 56-year-old female undergoing a bilateral iliac dilatation. The right iliac artery was successfully dilated and a left iliac artery dilatation was started in

TABLE II
ILIAC ARTERY LESIONS: RESULTS AND COMPLICATIONS

	Catheterization Attempted	*Inability To Cross Lesion*	*Balloon Dilatation Attempted*	*Initial Successful Dilatation (Of All Patients)*	*Poor Distal Run-off*	*Balloon Rupture*	*Distal Embolization*	*Distal Aortic Thrombosis*	*Vaso-Vagal Reaction*
Common or External Iliac Stenosis	63	3	60	55 (87.3%)	1	1	1	1	1
Common Iliac Occlusion	3	1	2	2 (66.7%)	0	0	0	0	0
Total	66	4	62	57 (86.4%)	1	1	1	1	1

an antegrade manner when the balloon ruptured near the bifurcation of the aorta. Several pieces were seen to go into the right as well as the left iliac artery under fluoroscopy. There was a complete occlusion of the right external iliac artery. Dilatation was attempted to relieve this obstruction of the right external iliac artery, but minimal distal flow was obtained. Following surgical consultation, an embolectomy was done on the right side. A bilateral aorto-femoral bypass was successfully performed later during the hospitalization.

A 49-year-old male undergoing bilateral common iliac dilatation had extreme stenosis of the common iliac arteries, starting at the bifurcation of the aorta. The left iliac artery was successfully dilated, but an attempt to dilate the right iliac artery resulted in poor distal run-off. Another attempt was being made to dilate the right iliac artery when an intraluminal thrombus was identified in the distal aorta at the bifurcation. The patient was taken to surgery for a right aorto-femoral bypass graft. At surgery, a large thrombus was identified at the distal aortic bifurcation. An embolectomy revealed thrombus in the right distal femoral artery as well. This was felt to be a fresh thrombosis, due to fibrin and platelet aggregation and not an atheromatous embolic phenomenon.

There was one complication secondary to warfarin (Coumadin) anticoagulation in a 63-year-old male who had recurrent bleeding from an old duodenal peptic ulcer site. He was treated with fresh frozen plasma, vitamin K, cimetidine (Tagamet), and discontinuation of the warfarin. One patient on dialysis developed a hematoma of the femoral artery entrance site (24 hours post PTA) during dialysis with heparin administration. He was treated with transfusion and conservative management without further complications. All of these patients are currently being followed for long term patency rates and complications to be reported in the future.

Discussion

The exact mechanism by which balloon dilatation exerts a remodeling influence on atheromatous plaque has been a much debated subject. From recent animal experiments and examination of human arteries at autopsy, several mechanisms of PTA success have been elucidated.[4, 5] First, PTA causes an increase in the outer diameter of the arterial wall, as well as the inner diameter. Second, histological studies of patients post successful PTA dying from an unrelated cause have shown superficial atheromatous desquamation and fragmentation. The dilated segment revealed vertical atheromatous plaque dissection up to the internal elastic lamina. Occasionally there is circumferential splitting also noted along the plane of the internal elastic lamina. Third, the damaged area undergoes inflammatory changes and subsequent fibrosis and retraction during a period of four to six weeks after angioplasty. Currently, more interest has been generated in the long term efficacy of this non-surgical procedure. Indeed, Dotter's first iliac balloon dilatation, performed 17 years ago, is patent today.[6]

The role of antiplatelet, anticoagulant, or fibrinolytic therapy in percutaneous transluminal angioplasty is yet to be clearly defined. As previously stated, we are currently using aspirin and dipyridamole (Persantine) for approximately six months post PTA. Technically, the angiographic and angioplasty catheters are undergoing marked improvement. The original polyvinyl chloride balloon catheters are being replaced by the newer polyethylene balloon catheters which are able to retain their original shape and withstand pressures up to 13-14 atmospheres without rupturing.[7]

Our results in the iliac vessels compare similarly to the work of others. We were most successful in treating short stenotic segment lesions. The work of others correlates with our initial success rate of 82-96.5 per cent in iliac stenosis, and 18-58.3 per cent for iliac occlusions.[8, 9, 10] Previously reported complications of PTA include distal embolization, acute thrombosis, vessel perforation, balloon rupture, and hematoma at the site of puncture.

Long term results of iliac artery lesions have been noted by Van Andel, who shows a 92 per cent patency rate in his group of 48 patients with 51 iliac artery lesions after a 33 month followup.[11] Spence *et al.*, have followed 148 iliac artery lesions for three years post PTA.[12] Of the successfully dilated iliac arteries, they have had one, two, and three year patency rates of 93.4, 87.2, and 79.4 per cent respectively. In comparing these results to surgery, Malone shows a 4 per cent thrombosis per year of followup in his aorto-femoral bypass series.[13] There is a serious complication in surgery of disruption of sympathetic innervation indispensible to sexual function in as high as 10 per cent of patients.[14, 15] Furthermore, it is difficult to compare the angioplasty patient population to the population undergoing surgical bypass grafting because the patients undergoing angioplasty are generally in worse medical shape and are often refractory to surgery in the first place. In the subgroup of patients who experience arterial restenosis after PTA, it is relatively easy to

TABLE III
COMPARISON BETWEEN ILIAC PTA AND TOTALLY UNCOMPLICATED AORTO-FEMORAL BYPASS

	Average Hospital Costs	*Average Hospital Stay (Days)*
Aorto-femoral Bypass, Totally Uncomplicated	$6,514	13.7
Iliac PTA	2,818	7.9
Savings	3,696 (56%)	5.9 (42%)

repeat angioplasty. PTA has the additional advantage of sparing the lower extremity veins for future harvesting for bypass grafting.

We conclude that PTA can be performed safely and effectively in the management of many patients with ischemic vascular disease. It appears that long term patency rates are acceptable and near comparable to surgery, although further followup is needed. With the current trend of increased cost consciousness in the field of health care, it is important to note Vawter's cost analysis between surgery and PTA as reviewed in *Table III*.[16] Patients undergoing PTA were able to cut 56 per cent of hospital costs, and 42 per cent of the hospital stay, as compared to a totally uncomplicated aorto-iliac bypass. PTA can also be used in conjunction with surgical therapy and should not be attempted without close cooperation between angiologist and vascular surgeon.[17] Long term results, possible improvement in sexual function, and the potential for outpatient utilization of this procedure must be further explored. In the future, we feel that PTA will be an important tool in the management of the patient with vascular disease, until the underlying etiology of atherosclerosis itself is delineated and becomes treatable.

Acknowledgement

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Pelvic Fractures

A Ten-Year Assessment

ROBERT P. BRUCE, B.S.,* *and* FREDERICK W. RECKLING, M.D.,† *Kansas City, Kansas*

FRACTURES of the pelvis are most commonly caused by automobile accidents and rank second only to skull fractures in mortality.[1] When treating a pelvic fracture, the physician must be aware of the potentially fatal complications associated with these injuries.

We have reviewed our experience with pelvic fractures at the University of Kansas Medical Center. The emphasis of our study is the correlation of specific types of pelvic fractures with the incidence of associated fractures and soft tissue injuries.

From January 1, 1971 to August 1, 1981, 142 patients with fractures of the pelvis were seen. Retrospectively, clinical records and radiographs were reviewed with 132 patients included in this study. Ten patients were eliminated due to inadequate documentation of injury or because they had sustained an avulsion fracture. Avulsion fractures were not included because they are almost always due to violent muscle contraction and rarely secondary to direct trauma. They seldom have associated complications.

Pelvic fractures were slightly more common in men (70 — 53%) than women (62 — 47%). Ages ranged from 4.5 years to 91 years. This is predominately a young person's injury, with 67 (51%) of the patients being between the ages of 15 and 30 years *(Table I)*. This is in sharp contrast to Peltier's study at this university covering the years from 1958 to 1963.[2] He found an even distribution of pelvic fractures throughout all decades of life. There were seven fatalities in our study, two of which were fetal deaths.

Automobile accidents were the leading cause of injury accounting for 52 per cent of cases. Pedestrians hit by an automobile were second in frequency. Thus, two-thirds (67%) of all pelvic fractures were automobile related injuries. In patients over 60 years of age, a fall in the home was most often the cause of the fracture, ranking third overall. This is similar to the findings of Hauser and Perry.[3]

A classification scheme must be adopted to properly assimilate injuries associated with pelvic fractures. We have classified pelvic fractures into two large categories: those that affect weight bearing structures and those that affect non-weight bearing structures. This was described by Peltier[2] *(Table II)*.

A study of pelvic fractures treated at UKSM-KC during 1971-1981 shows markedly improved results over cases treated there 1958-1963. Overall, results were also superior to those reported in other studies.

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TABLE I
AGE DISTRIBUTION OF PATIENTS BY DECADE

Age in Years	0-10	4	41-50	15
	11-20	35	51-60	15
	21-30	38	61-70	8
	31-40	10	71 & over	7

Number of patients between age 15 & 30 = 67 (51%)

TABLE II
CLASSIFICATION OF FRACTURES OF THE PELVIS

I. Fractures not affecting weight-bearing
- Avulsion fractures
- Isolated fractures of the wing of the ilium (Duverney)
- Fractures of the pubic rami
 - Unilateral
 - Bilateral

II. Fractures affecting weight-bearing
- Isolated fractures of the sacrum
- Fractures of the acetabulum
- Separation of the symphysis
- Fractures of the hemipelvis (Malgaigne)

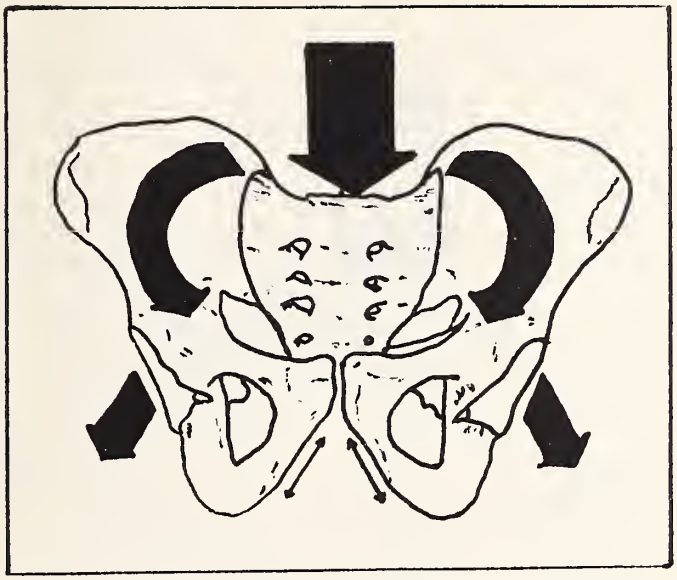


Figure 1. Weight bearing force transmission through the pelvis.

Weight bearing forces are transmitted from the vertebral column to the sacrum, through the sacroiliac joints and ilia, to the acetabula, and finally through the femora as shown in *Figure 1*. Thus the anterior one-third of the pelvis is not involved in weight bearing. Fractures through this area are of little consequence as far as ambulation is concerned.

The frequency and variety of associated fractures in our series is shown in *Table III*. *Table IV* depicts the number of associated soft tissue injuries. The large number of associated injuries emphasizes the care with which the physician must examine patients with suspected pelvic fractures.

Duverney[4] first described an isolated fracture of the wing of the ilium in 1751 *(Figure 2)*. These are caused by direct blows with lateral compression forces. We treated only three such cases (2.2%); all of these had associated injuries. There were no visceral injuries. All responded well to bedrest and gradual ambulation.

Unilateral fractures of the pubic rami *(Figure 3)* are the most common fractures of the pelvis. They comprised 44 patients (34%) of our series including one fatality. Seventeen (38%) sustained associated injuries; 13 of these were fractures of the lower extremity, the tibia or fibula being the most commonly fractured bones. Although the x-ray appearance of the pelvic injury may seem benign, one patient had a pneumothorax, two had hemothoraces, two had ruptured spleens, and one had a urethral tear. The fatality was secondary to gram negative sepsis from multiple facial fractures and was not directly related to the pelvic fracture. This is also the

TABLE IV
SOFT-TISSUE INJURIES ASSOCIATED WITH FRACTURES OF THE PELVIS IN 132 PATIENTS

Brain Injury	6	Ruptured Ilium	2
		Ruptured Colon	1
Pneumothorax	5	Mesenteric Tear	4
Hemothorax	5		
Ruptured Diaphragm	1	Ruptured Bladder	6
Aortic Tear	1	Urethral Injury	5
Ruptured Spleen	7	Lumbar-Sacral	
Laceration of Liver	2	Plexus Injury	1
Transection of Pancreas	1		

TABLE III
FRACTURES ASSOCIATED WITH PELVIC FRACTURES IN 132 PATIENTS

	Fractures		
Skull	6	Ribs-Sternum	23
Facial Bones	16	Vertebrae	24
Clavicle	4	Femur	26
Scapula	2	Patella	2
Humerus	5	Tibia-Fibula	31
Radius-Ulna	10	Tarsal-Metatarsal	4
Carpal-Metacarpal	3		
	Dislocations		
Hip	13	Knee	1
Shoulder	2	Elbow	1

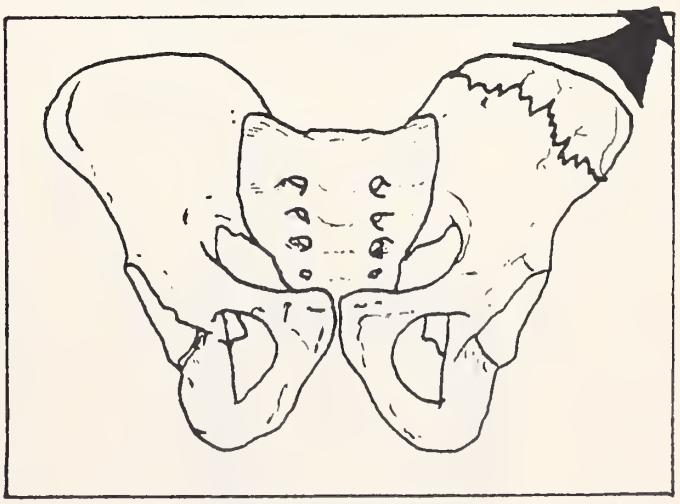


Figure 2. Isolated fracture of the wing of the ileum (Duverney).

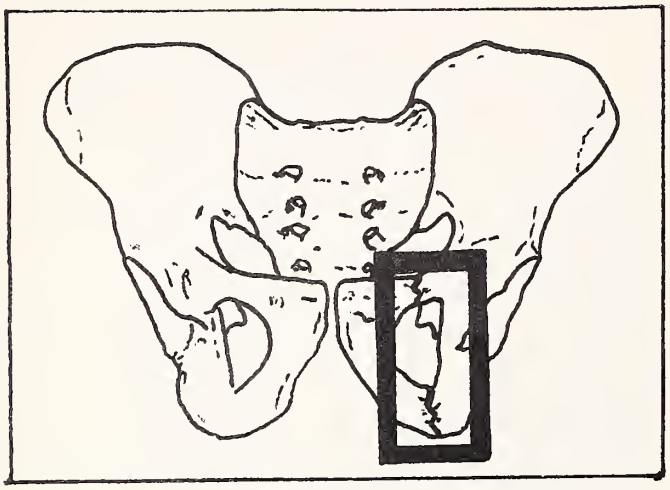


Figure 3. Unilateral fractures of the pubic rami.

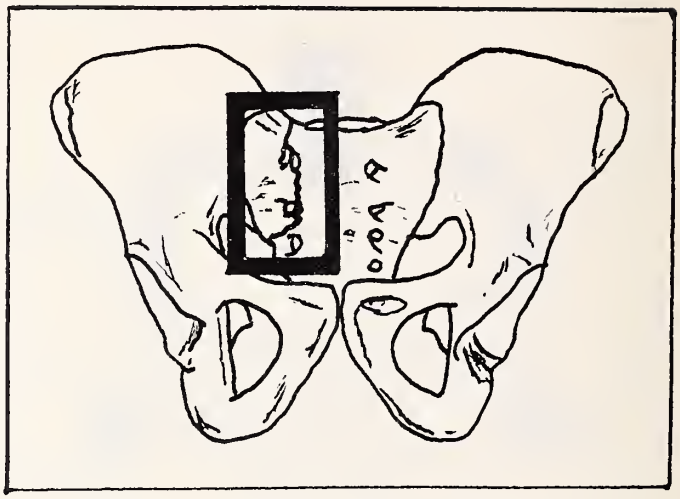


Figure 5. Isolated fracture of the sacrum.

most common pelvic fracture sustained in falls. Eleven (24%) of the total occurred in this manner. The majority of these patients are over the age of 60 years. Patients with this injury were hospitalized for an average of 14 days.

Bilateral fractures of the pubic rami *(Figure 4)* occurred in 20 patients (15%) in this series. Eighteen (90%) had other injuries and there was one fetal death. The fetus was 38 weeks gestation and the pelvic fracture was the mother's sole injury from which she recovered without difficulty. There were three visceral injuries (15%) including one ruptured spleen, one mesenteric tear, one ruptured bladder, as well as one urethral tear. This differs from Peltier's[2] experience in which he had an incidence of visceral injuries of 39 per cent and a mortality rate of 19 per cent. Likewise Conolly and Hedberg[1] had a higher incidence of associated lower urinary tract damage (35%) with bilateral pubic rami fractures.

In our limited series, we did not encounter any isolated fractures of the sacrum *(Figure 5)*.

Fractures of the acetabulum *(Figure 6)* accounted for 31 (24%) of patients. Nine of these (7%) had central fracture-dislocation of the hip as shown in *Table V*. These are more severe injuries and will be considered separately. Of the remaining 22 patients, 14 had associated injuries and there was one fetal death. This fetus was also in its last month of gestation at the time of accident. All of these 14 patients sustained other fractures with the facial bones most commonly fractured. There was only one visceral injury — a mesenteric tear — in this group and there were no urinary tract injuries. Five fractures of the acetabulum required open reduction and internal fixation for stabilization.

Of the nine patients with central fracture-dislocations of the hip, all sustained associated injuries (100%). Six of nine had fractures of the lower

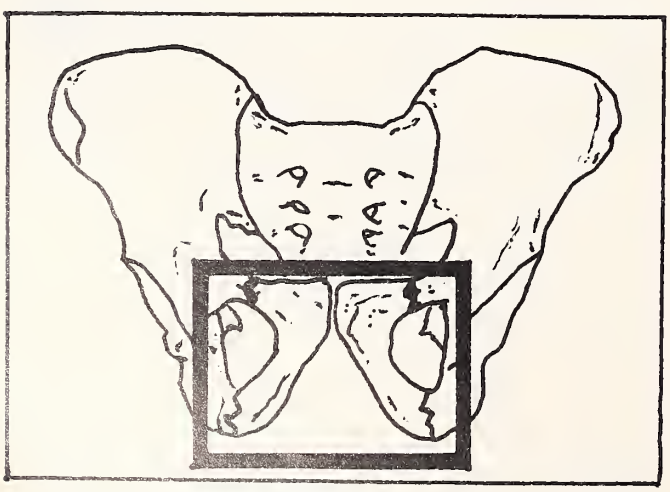


Figure 4. Bilateral fractures of the pubic rami.

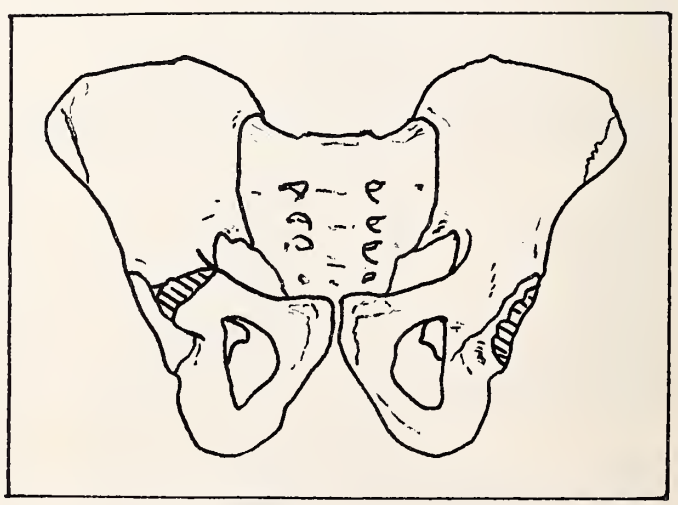


Figure 6. Fractures of the acetabulum.

TABLE V

Type of Fracture	*No. (per cent)*	*Morbidity*	*Mortality*	*Visceral Injuries*	*Fractures of Lower Extremity*	*Lower Urinary Tract Injuries*
Duverney	3 (2%)	3 (100%)	0	0	1 (33%)	0
Unilateral pubic rami	45 (34%)	17 (38%)	1 (2.2%)	2 (4.4%)	13 (29%)	1
Bilateral pubic rami	20 (15%)	18 (90%)	1 fetal death	3 (15%)	5 (25%)	2
Isolated fractures of the sacrum	0	0	0	0	0	0
Fractures of the acetabulum	22 (17%)	14 (64%)	1 fetal death	1 (4.5%)	6 (27%)	0
Central fracture — dislocations of the hip	9 (7%)	9 (100%)	0	3 (33%)	6 (67%)	1
Separation of the symphysis	3 (2%)	3 (100%)	0	0	1 (33%)	1
Fractures of the hemipelvis (Malgaigne)	30 (23%)	29 (97%)	4 (13%)	9 (30%)	13 (43%)	6
Total	132	93 (70%)	5 (3.8%) 2 fetal deaths	18 (14%)	45 (34%)	11 (8.3%)

extremity. There were three visceral injuries with two ruptured spleens and one ruptured bladder. In one case, the bladder was entrapped by the fracture fragments requiring open reduction. Three additional patients required open reduction and internal fixation. One patient had fat embolus.

Three patients had separation of the symphysis pubis *(Figure 7)*. Two of them were caused by motorcycle accidents. All had associated fractures. There were no visceral injuries but there was one ruptured urethra. One patient was treated with a Hoffman external fixator for 90 days and had excellent final result.

Fracture of the hemipelvis *(Figure 8)* was first described by Malgaigne in 1847.[5] This fracture is a double verticle break in the pelvic ring with the anterior portion being a break in the superior and inferior pubic rami or a separation of the symphysis pubis. The posterior portion is composed of a break behind the acetabulum or a fracture-dislocation of the sacroiliac joint. The intervening segment of the pelvis must also be displaced. We had 30 such injuries (23%) in our series. The incidence of this fracture appears to be increasing. Peltier[2] reported an overall incidence of 7 per cent. Twenty-nine of the 30 (97%) with Malgaigne fractures had associated injuries. Thirteen of 29 sustained vertebral fractures, and an equal number sustained fractures of the

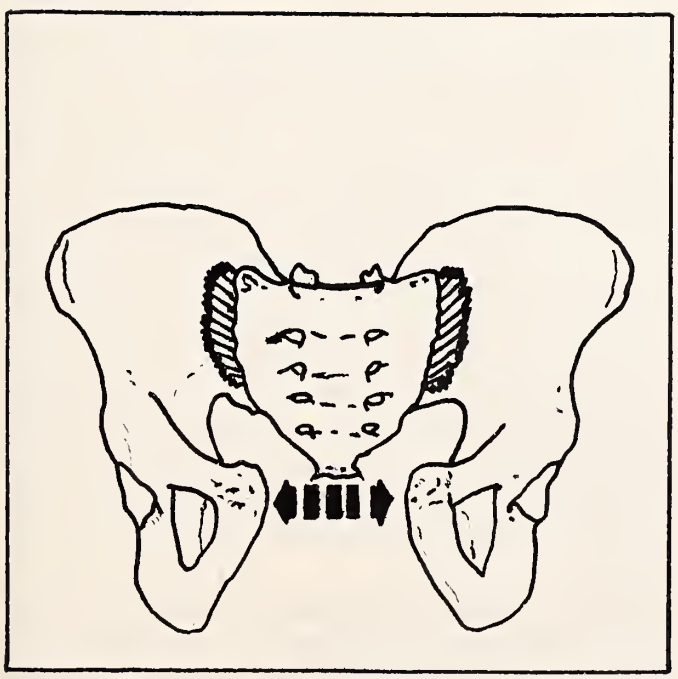


Figure 7. Separation of the symphysis pubis.

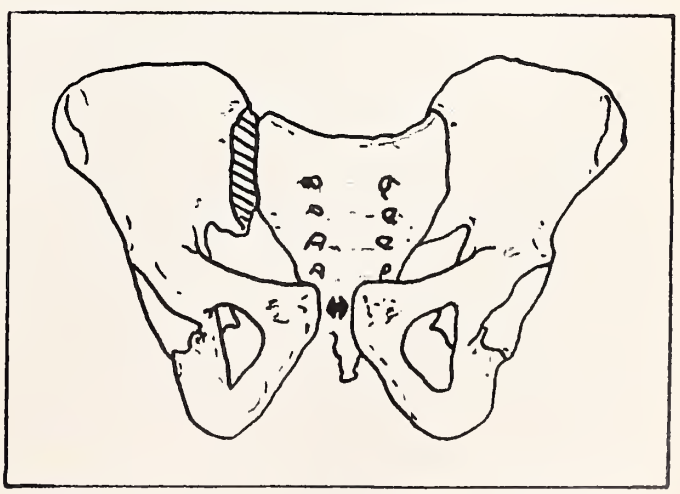


Figure 8. Fracture of the hemipelvis (Malgaigne).

lower extremity. The Malgaigne fracture had the highest incidence of associated visceral injuries. There were three ruptured spleens, two mesenteric tears, and four ruptured bladders discovered during 12 exploratory laparotomies. Seven patients developed fat emboli with four fatalities (a mortality rate of 13%). Two patients were treated with open reduction and internal fixation, and one with a Hoffman external fixator. The average hospital stay for patients with this fracture was 37 days.

Of the four fatalities, only one died of causes attributable to the pelvic fracture itself. This patient died on the third hospital day of renal failure secondary to shock from massive intraperitoneal bleeding. One patient died of bacterial and fungal encephalitis 22 days after injury. This patient sustained an open fracture of the hemipelvis with an accompanying sacral fracture and a ruptured colon. Feces contaminated the meninges before a colostomy could be performed. Another patient died of complications from a head injury after 33 days. One patient died 64 days after initial hospitalization and the cause of death was not determined.

Discussion

Of 132 patients with pelvic fractures, 70 per cent had associated injuries. Five (3.8%) of the patients had fatal complications. This is one of the lowest mortality rates reported in recent literature. Peltier[2] cited a 9 per cent mortality rate in 1965; Dunn and Morris[6] reported 5.2 per cent; Conolly and Hedberg,[1] 15 per cent; and Trunkey[7] found a rate of 9.2 per cent in 1974. There are several possible explanations for this finding. One is that our series was composed of many relatively mild pelvic fractures with less intraperitoneal bleeding, fewer incidences of visceral injury, and other potentially fatal complications.

Bilateral fractures of the pubic rami, central fracture-dislocations of the hip, and fractures of the hemipelvis (Malgaigne) are cited in other studies for a high incidence of mortality and morbidity. These fractures accounted for 59 (45%) of our series.

Intrapelvic hemorrhage is perhaps the most serious complication of pelvic fractures. Hauser and Perry[3] had 15 deaths in their series of 196 patients, nine of which were due to hemorrhage alone. Conolly and Hedberg[1] determined that 75 per cent of the fatalities in their study of 200 pelvic fractures were due to hemorrhagic shock or renal failure secondary to shock. This was not found to be a major factor in the fatalities in our series.

We feel that very early blood volume replacement and pelvic fracture stabilization to prevent further hemorrhage are the keys to lowering mortalities from this injury. Constant monitoring of central venous pressure and urinary output is necessary for maintaining intravascular volume. Pelvic or retroperitoneal hematomas which commonly accompany pelvic fractures may hold as much as 8-12 units of blood. Cystograms are useful not only in evaluating lower urinary tract injuries but also in estimating the size of the hematomas. Peltier[2] believes as we do that all patients with pelvic fractures should have retrograde cystograms for this reason whether or not a urologic injury is suspected.

Prompt reduction and immobilization of unstable pelvic fractures will help to tamponade bleeding from bone ends and small veins — the most common source of hematomata. This may be done with a variety of methods including Buck's traction with a pelvic sling, plaster spica casts, or with internal or external fixation.

Almost one-half (48%) of those patients with associated injuries sustained a fracture of the lower extremity *(Table V)*. This occurred most commonly with central fracture-dislocations of the hip and the Malgaigne fracture. Fractures of the lower extremity can also contribute greatly to blood loss thus compounding the loss created by the pelvic fracture. The physician must not ascribe early signs of hemorrhage to this injury and ignore the potentially greater loss of blood in the pelvis.

External skeletal fixation provides an excellent alternative treatment for patients with disruption of both the anterior and posterior portion of the pelvic ring. It provides accurate reduction that is rigidly maintained allowing early mobilization, ease of nursing care, and considerable patient comfort. We used a Hoffman apparatus attached to six pins inserted in each iliac crest. With adjustment of the slider bar, complete reduction was achieved by compressing the sacroiliac joints as well as the symphysis pubis. The combination of anterior and posterior compression offers a distinct advantage over other methods of fixation such as wiring or nailing of the symphysis. About two weeks after application, consideration can be given to ambulation. Prior to this time however, active range of motion physiotherapy can be given to prevent deep venous thrombosis. Twelve weeks post-fracture the apparatus is removed. In our patients anatomic reduction was maintained throughout and following removal of the fixator.

Eight per cent of our patients had urologic injuries. Morehouse *et al.*[8] found an incidence of 10 per cent in his study of pelvic fractures. Urethral ruptures tend to be more common than ruptures of

the bladder,[9] although we found them to be essentially equal with six ruptured bladders and five urethral tears. Some authors have found that the likelihood of urologic injury is related to the number of pubic rami fractured.[9] We found more than one-half of these injuries associated with the Malgaigne fracture.

Hematuria is a common presenting sign in patients with fractures of the pelvis. As shown in *Table VI*, 47 per cent of our patients were admitted with either microscopic or gross hematuria. After retrograde cystogram, 11 were found to have urologic injuries. Thus, only 18 per cent of the patients with hematuria had significant urologic injuries. Hematuria in the remaining patients was most likely due to bladder mucosa contusion. Although there is a low percentage of urinary tract injuries in those patients with hematuria, all demand evaluation. As Bright *et al.*[10] found in their study of 142 trauma patients, the amount of hematuria does not correlate with the presence or absence of urologic injury. Conolly and Hedberg[1] also found that hematuria itself is not indicative of significant injury to the urinary tract.

The only two women who were pregnant at the time of their pelvic fractures aborted. Both were near term. One sustained bilateral fractures of the pubic rami and the other a fracture of the acetabulum. It is obvious that forces transmitted to the pelvis sufficient to cause a fracture may be lethal to the fetus. Speer[11] found that 34 per cent of women pregnant at the time of pelvic fracture suffered fetal loss.

TABLE VI
HEMATURIA AND UROLOGIC INJURY IN 132 PATIENTS WITH PELVIC FRACTURES

Hematuria	*Number of Patients*
Microscopic	28
Gross	34
Total	62

Incidence of Hematuria = 47%

Type of Fracture	*Ruptured Bladder*	*Urethral Tear*
Unilateral pubic rami		1
Bilateral pubic rami	1	1
Central fracture — dislocation of hip	1	
Separation of symphysis		1
Fracture of the hemipelvis	4	2
Total	6	5

Incidence of urologic injury = 8.3%
Incidence of urologic injury in patients with hematuria = 18%.

The incidence of significant urologic injuries was 8.3 per cent. The presence of hematuria may not be a reliable sign of significant lower urinary tract injury, but must be investigated in all instances.

External skeletal fixation via the Hoffman apparatus is a viable alternative to conventional treatment of pelvic ring disruption.

Summary

A series of 132 pelvic fractures was reviewed. Automobiles were responsible for causing two-thirds of the fractures in this predominately young population.

The fractures were classified as weight bearing and non-weight bearing. Unilateral fractures of the pubic rami are the most common pelvic fractures. Seventy per cent of all the patients had associated injuries with a fracture of the lower extremities occurring most often. The Malgaigne fracture is becoming more common. Malgaigne fractures comprised one-fourth of our total series and had the highest incidence of visceral and urologic injuries as well as the highest mortality rate. This probably occurs as a result of increasing numbers of rapid velocity injuries.

The mortality rate in this series of 3.8 per cent is one of the lowest reported. We believe some of this was attributable to rapid blood volume replacement and early pelvic fracture stabilization.

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Prostatic Carcinoma

Interstitial Radiation Therapy

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CARCINOMA of the prostate is second only to lung carcinoma as the most common malignancy in men. It usually affects men over the age of 50 years, with the majority of cases occurring in the sixth and seventh decades of life. The most common symptoms are urinary hesitancy, increased frequency of urination, and nocturia. Carcinoma of the prostate usually arises in the peripheral zone of the prostate, and it is generally easy to palpate on digital rectal examination. Any size nodule in the prostate in a man over the age of 50 years is considered to be malignant until proven otherwise.

An accurate staging is most important as it relates to both treatment and prognosis. Staging procedures, different staging systems, and histology and grading have been described previously.[1] Although TNM staging classification is available, most clinicians in the United States use Whitmore[2] or Jewett[3] staging systems:

Stage:	A_1	Focal — well differentiated
	A_2	Multifocal — anaplastic
	B_1	< 1.5 cm nodule
	B_2	> 1.5 cm nodule
	C	Extracapsular extension
	D	Metastasis

Treatment

More than 50 per cent of patients with the diagnosis of carcinoma of the prostate present with distant metastases at the initial presentation. About 35-40 per cent of patients present with locally advanced disease. Only 10 per cent or less of patients present with truly localized disease, such as stages A or B_1. Radical surgical approach can be utilized only in such localized cases, but with high morbidity manifested by urinary incontinence and impotency. Since the advent of megavoltage radiation equipment, radiation therapy is playing a major role in the management of carcinoma of the prostate. The primary advantage of megavoltage radiation — such as cobalt and high energy linear accelerators — is that a high tumoricidal dose to the prostate can be delivered without undue damage to normal structures such as skin and subcutaneous tissues. Radiation therapy in the curative management of carcinoma of the prostate can be carried out by two methods: external radiation therapy or interstitial radiation. The application of external radiation therapy has been described previously.[1] This paper will deal with combined surgical and interstitial radiation treatment of carcinoma of the prostate.

Radiation therapy is playing a major role in the management of carcinoma of the prostate. This is usually done by external radiation therapy or interstitial iodine-125 radioisotope implant of the prostate in conjunction with bilateral pelvic lymphadenectomy. The combined surgical and implantation technique offers certain advantages, but it has not yet been shown that results are any better in terms of local control or survival than those of external radiation therapy.

Interstitial Radiation

Pasteau and Degrais[4] used intraurethral radium to treat carcinoma of the prostate as early as 1909. In 1915, Barringer[5] advocated interstitial implantation, using radium needles and gold seed implants, and showed the ability of ionizing radiation to sterilize prostatic cancer in four patients treated by the above method. In 1952, Flocks[6] used a method of instillation of radioactive colloidal gold directly into the prostate gland or into the tumor bed after subtotal resection. The main disadvantages of the above methods are undue side effects to the patient and radiation exposure to personnel. In 1970, Whitmore and Hilaris[7] used a new radioisotope — iodine-125 — as a substitute for gold and radon seeds in con-

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TABLE I
RADIONUCLIDES FOR INTERSTITIAL IMPLANTS

Isotope	*Half Life (Days)*	*Emitted Energy* *Alpha*	*Beta*	*Gamma*	*Half Value Layer (Lead)*	*Half Value Layer (Tissue)*
Iodine-125	60	None	None	27-35 keV	0.025 mm	2 cm
Iridium-192	74.4	None	670 keV	300-610 keV	4.5 mm	6 cm
Gold-198	2.7	None	960 keV	410 keV	4.5 mm	6 cm
Radon-222	3.8	+	70-3260	50-2440 keV	13 mm	10 cm

junction with bilateral pelvic lymphadenectomy as a staging procedure.

Iodine-125 has some practical and theoretical advantages over other radioisotopes *(Table I)* with a long half life of 60 days and a peak energy of 31 keV. The intensity of gamma radiations is reduced to half by two cm of soft tissue, while 8-10 cm of tissue will reduce the activity to 3 per cent. Kim and Hilaris[8] showed that iodine-125 has a greater therapeutic ratio — *i.e.* equal or better local control rate with minimal complications — when compared to radon 222 and gold 198. Because of low gamma energy, the radiation exposure is negligible to personnel handling the isotope in the operating room, and the patient need not be put in isolation following the procedure. There are no special precautions to be observed by the patient or the family.

All patients with the diagnosis of localized carcinoma (A, B, C) of the prostate who are in reasonably good health with an estimated life expectancy of five or more years are eligible for this combined surgical and radiotherapeutic procedure. The only exceptions are bulky stage C lesions with tumor extension to the pelvic wall or bladder base because it is difficult to get a uniform distribution of the isotope. Patients presenting with obstructive symptoms pose a special problem because these patients require transurethral resection to relieve the obstruction and also to prevent total obstruction from prostate swelling from the radioisotope implant. A minimal or conservative transurethral resection of the prostate (TURP) is advised in these patients because total TURP will leave no tissue to hold the implant. A waiting period of three to five weeks is essential to permit healing

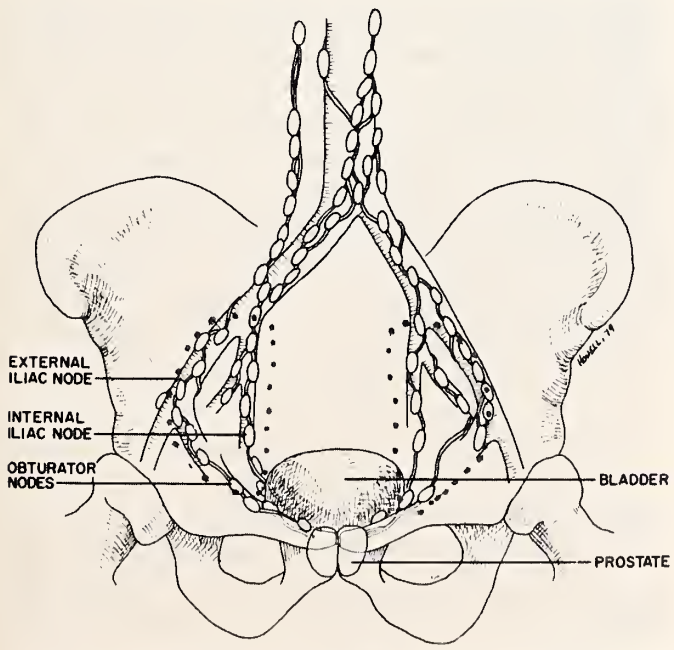


Figure 1. Lymphatic drainage of prostate. The nodes within dotted lines removed during surgery.

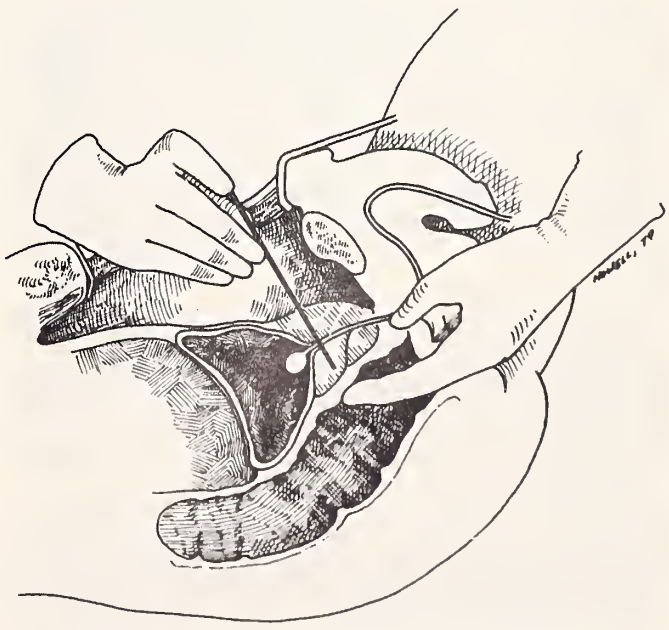


Figure 2. Technique of insertion of needles.



Figure 3a. Anterior localization film showing the seeds.



Figure 3b. Lateral localization film with contrast in the rectum.

and resolution of infection before the implantation can be carried out. After bilateral pelvic lymphadenectomy *(Figure 1)* and prostatic exposure, the implantation of the entire gland is carried out by using 15 cm long hollow 17 gauge stainless steel needles. The needles are placed starting from base to apex, avoiding the median plane to prevent the insertion of needles into the urethra. The needles are inserted in more or less anterior-posterior direction parallel to one another approximately 1 cm apart, guiding the needles with the help of the index finger in the rectum *(Figure 2)*. When the insertion of the needles is completed, the optimal activity (millicuries) of I-125 required for the implant is determined utilizing the "average dimension" method[9] as follows:

A = da × K
A = total activity in millicuries required
da = dimension average (length + width + depth/3)
K = constant of proportionality, *e.g.*, '5' for iodine-125

The total millicuries, obtained by the average dimension method, divided by the average activity of the iodine-125 seeds available (usual activity per seed is about 0.5 millicuries), yields the actual number of seeds required. A semi-automatic applicator which uses magazines holding 14 seeds each, is utilized to deposit the seeds at 1 cm intervals as each needle is successively withdrawn. Prior to actual loading of the needles with iodine-125 seeds, it is important to withdraw the tip of the needle by at least 0.5 cm, to avoid placing the seed close to the rectal wall. When the patient has recovered from surgery, on the fourth or fifth postoperative day, localization films of the implant are done *(Figure 3)*. From these, a computer generated isodose distribution is plotted *(Figure 4)*. The total activity required for said tumor volume will deliver about 16,000-18,000 rad at total decay of the isotope, which is about one year. With pelvic lymphadenectomy and I-125 implant of the prostate, intra-operative complication was 6 per cent,[10] and 23-34 per cent[10, 11] had postoperative complications. About 50 per cent of patients developed voiding symptoms following the procedure, and a majority of them had subsidence of symptoms by one year.[10] The reported frequency of impotency with interstitial radiation is 7-12 per cent.[10, 12]

Results

With bilateral pelvic lymphadenectomy and iodine-125 implant of the prostate, Hilaris *et al.*[13] achieved a 73 per cent overall survival rate. The survival rate is lower with advanced stage carcinoma. When the lymph nodes were histologically negative, 92 per cent of patients survived for five years or more, compared to 46 per cent with positive nodes. At the University of Kansas Medical Center,

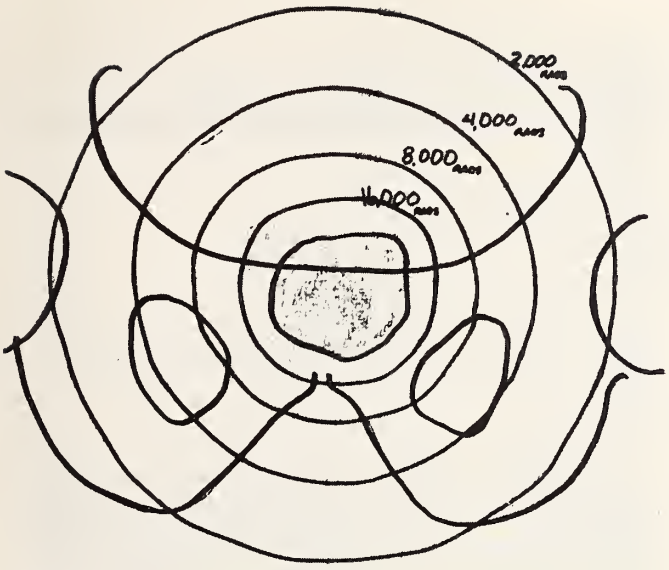


Figure 4a. Computer generated isodose distribution (anterior).

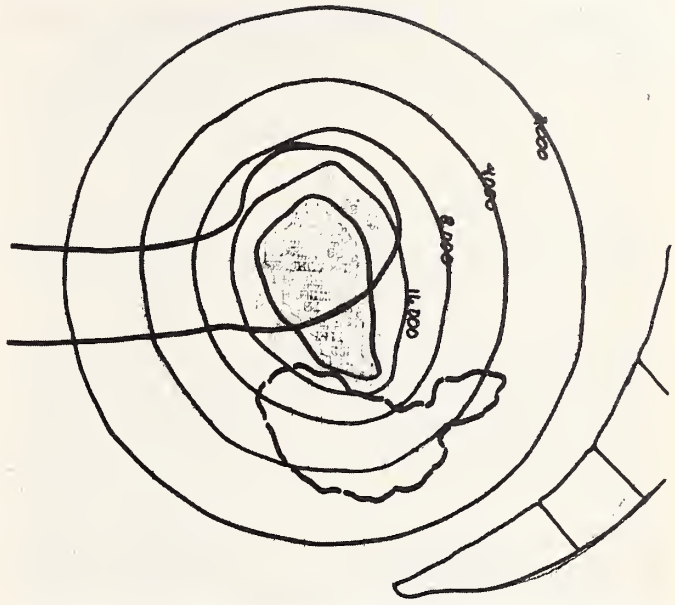


Figure 4b. Computer generated isodose distribution (lateral). 16,000 rad isodose line represents the minimum tumor dose.

88 patients underwent this combined procedure from 1976-1981, and 60 patients had a minimum of two years followup. Only one patient died of distant disease and five patients are living with distant metastases. All of these five patients had positive nodes at the time of surgery.

The advantages of this procedure over external radiation therapy are the shorter treatment time (external radiation therapy takes 7-8 weeks), more accurate staging, and a lower incidence of sexual impotency. The radioactive seeds may be precisely placed into the neoplastic prostate and a high dose of radiation can be delivered to the prostate. It is a treatment option available for suitable candidates. The main disadvantage is the associated surgical procedure with its attendant mortality and morbidity. It requires a long term followup of 10-15 years to determine the efficacy of any treatment in carcinoma of the prostate. Although the preliminary results of this procedure by Hilaris *et al.*[13] suggest a favorable outcome, the ultimate role of this procedure is yet to be determined. At present, interstitial radiation is not superior to external radiation therapy in achieving significantly better local control or survival.

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Renal Transplantation

A Six-Year Experience With Children and Adolescents

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TREATMENT of end-stage renal disease during the last decade has progressed sufficiently so that dialysis and transplantation are now technically feasible in children and can lead to excellent long-term results.[1] However, since long term dialysis is not suitable for children, the emphasis has been placed on renal transplantation as the preferred mode of treatment of end-stage renal disease in childhood.[2, 3]

Here we briefly review the present status of renal transplantation in children and summarize our personal experience with 12 kidney transplants performed in 11 pediatric patients between 1976 and 1982 at the University of Kansas Medical Center.

Results

The results of our experience with dialysis and transplantation in children are presented in *Tables I* and *II*. The patients ranged in age from 5-15 years and all received cadaver allografts. The smallest patient weighed 12 kgs. The donors ranged in age from 2-41 years. Generally, we selected cadaveric kidneys from young donors for smaller patients but no problems occurred from grafting adult kidneys into small patients. Followup on the 11 patients varies between three months and six years.

Eleven of our patients had either congenital or hereditary renal disease. Only one patient (RA) suffered from an acquired glomerulopathy.

All but two of the 11 patients were treated with either peritoneal or hemodialysis. Two patients (SB and KA) received kidney transplants shortly before they were to begin dialysis.

We have had wide variability in the degree of HLA, A, B, C, and Dr matching of our patients, and at least one patient (SC) received only a blood group compatible kidney with no other matching tissue antigen. Since 1980, we have given blood transfusions routinely to all patients prior to transplantation.

Twelve kidney transplants were performed in 11 pediatric patients at UKSM-KC between 1976 and 1982. From the literature and the results of our experience, we conclude that — despite many drawbacks and remaining problems — the present treatment of end-stage renal disease in children can produce satisfactory results.

Our antirejection protocol was as follows: All patients received intravenous cyclophosphamide and methylprednisolone at the time of transplant surgery and during the next five days. Patients were then maintained on azathioprine 2-2½ mg/kg/day and prednisone. The starting dose of prednisone was 2 mg/kg/day, and was reduced to 0.5 mg/kg/day during the next two to three weeks. This daily prednisone schedule was then gradually converted to a dose of 0.5 mg/kg/ given every other day. Despite the antirejection therapy, all patients had at least one episode of acute rejection during the first two months following transplantation. These episodes of rejection were treated with intravenous injections of methylprednisolone (30 mg/kg) to a maximum of one gram daily for four to nine days. One of the patients (JS) underwent an accelerated rejection and despite aggressive antirejection therapy, lost the transplant after three weeks. The patient returned to hemodialysis and received a second renal allograft 15 months later which also underwent accelerated rejection. This time, we gave the usual antirejection therapy in combination with plasmapheresis, and the rejection process abated. No rejection episode has occurred since.

Following transplantation, all patients had some degree of hypertension. Eight patients had mild to moderate elevation of blood pressure, but in four patients hypertension could only be controlled after bilateral nephrectomy. Six patients demonstrated significant growth failure prior to transplantation

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TABLE I
PATIENTS TRANSPLANTED (CADAVERIC ALLOGRAFTS)
DEPARTMENT OF PEDIATRICS, UNIVERSITY OF KANSAS MEDICAL CENTER 1976-1982

Patient	*Age (yrs)*	*Sex*	*Race*	*Weight (kg)*	*Height (cm)*	*Height %*	*Dialysis*	*Diagnosis*
JA*	12 3/12	F	W	38	156.5	25	Peritoneal	Nephronophthisis
MF	13 11/12	F	W	39	154.1	25	Peritoneal	Pyelonephritis
RP	7 9/12	M	W	13	104	<5	Peritoneal	Constricting thoracic dystrophy sclerosing glomerulopathy
SC	7 8/12	F	W	13	111.0	<5	Peritoneal	Hypoplastic kidneys
RA	16 3/12	M	W	28	137.9	<5	Peritoneal-Hemo	Focal segmental sclerosis
JS†	8 7/12	M	W	26	132.5	50-75	Hemo	Obstructive uropathy
	9 10/12			29	136.5	50	Hemo	
JP	12 9/12	F	W	31	132.0	<5	Peritoneal	Nephrosclerosis
JW	12 6/12	F	W	22.5	122.3	<5	Peritoneal	Hypoplastic kidneys
KA*	15 3/12	M	W	52	170	50	None	Nephronophthisis
AR	9 7/12	F	B	20	121.5	<5	Hemo	Unknown
SB	5 7/12	M	W	12	93	<5	None	Hypoplastic kidneys

* Siblings.
† Two Transplants.

TABLE II
TRANSPLANTATION IN CHILDREN
OUTCOME

Patient	*Date of Transplant*	*Complications*	*Clinical*	*Serum Creatinine (mg/dl)*
JA	7-76	Bone demineralization	Well	0.9
MF	10-77	Bone demineralization Hypertension Hyperglycemia	Well	1.0
RP	7-79	Growth retardation Hypertension	Well	0.7-0.9
SC	11-79	Growth retardation Cataracts Hypertension CVM Hepatitis	Well	0.7
RA	5-80	Growth retardation Hypertension Seizures	Proteinuria Hematuria	0.7
JS	8-80 11-81	Accelerated rejection Severe rejection	Nephrectomy Well	0.6-0.8
JP	2-81	Growth retardation Hypertension Delayed puberty	Well	0.8
JW	4-81	Growth retardation Hypertension Decreased renal function	Well	1.3
KA	6-81	Hypertension	Well	
AR	2-82	Growth retardation Hypertension Necrosis of ureter Herpes simplex conjunctivitis	Well	0.6
SB	2-82	Growth retardation	Well	0.4

and were below the fifth percentile for height and weight. None of these patients has demonstrated significant catch-up growth. Two patients show significant bone demineralization, probably secondary to steroid therapy, but neither has had a spontaneous fracture or signs of aseptic necrosis. Three patients needed temporary dialysis post-transplantation because of delayed graft function. All patients have presently functioning grafts; function is slightly decreased in only one (JW). Our overall results are exceptionally good, and we have encountered only one postoperative surgical complication. AR developed urinary extravasation secondary to partial necrosis of the ureter at the site of reimplantation which required surgical repair.

Discussion

In every patient with progressive renal disease, a point will be reached at which conservative treatment consisting of diet, maintenance of water and acid-base balance, and medication will fail. Dialysis is generally initiated when renal function has deteriorated to a point where creatinine clearance is less than 5 ml/min/1.73sq M body surface area, or the patient develops significant uremic symptoms, especially progressive bone disease, enteritis, and cardiac and peripheral nerve involvement.

The age and size of patients are no longer limiting factors for dialysis and transplantation. In many centers, children weighing as little as 10 kg are candidates for both modes of therapy.[2] Many pediatric nephrologists favor early transplantation perhaps even prior to initiating dialysis, thus eliminating this traumatic experience.

All of our patients have received cadaveric allografts because the family situations have not lent themselves to providing a live-related donor kidney. In other centers, live-related donors are preferred because of a somewhat better graft survival.[4]

Although some care must be given to the size of the donor kidney, adult kidneys can be transplanted into young children and conversely, adult patients have received pediatric grafts.[5] If possible, an attempt is made to transplant the kidney extraperitoneally. When a small kidney is transplanted into a larger recipient, the kidney hypertrophies and will eventually provide adequate renal function.

Since obstructive uropathy is a common cause of end-stage renal disease in children, provision for adequate drainage of the lower urinary tract has to be made prior to transplantation. When the lower urinary tract is abnormal, the transplanted kidney can be drained into a previously constructed colon conduit, as was accomplished in JS.[6]

Despite many advances in the field of transplant immunology, the cornerstone of antirejection therapy is still medical treatment with a combination of corticosteroids and azathioprine. Various other therapeutic regimens have been proposed but with the exception of pretransplant blood transfusions, there has been little advancement in this area in the past ten years.

A significant increase in graft survival has been noted to occur in patients who received blood transfusions prior to renal transplantation. The reason for enhanced graft survival after blood transfusion is not clear, but the clinical results are unequivocal.[7] Beginning with patient RA, we have made a concerted effort to see that all of our transplant patients receive at least three to five transfusions with buffy coat poor washed packed red blood celis. Another factor involved in graft survival may be the histocompatibility antigens.[8] In the present series, there was no consistent degree of matching for HLA, A, B, and C or Dr antigens in our patients, and we have seen no relationship between graft survival and HLA, A, B, and C matching.

Following transplantation, patients are monitored carefully for four to six weeks for evidence of acute rejection. Several factors are helpful in diagnosing acute rejection including decreasing urinary output, increasing blood pressure, presence of fever and graft tenderness, leukocytosis, thrombocytopenia, an elevation of serum creatinine, alterations in fractional renal sodium excretion, and the presence of fibrin split products in the urine. We also utilize sonographic and radioisotope studies of the kidney. Episodes of transplant rejection are treated with pharmacologic doses of methylprednisolone given daily and intravenously. In some centers, renal biopsy is also used to help diagnose rejection episodes. However, it has been our experience that with careful clinical and laboratory follow-up, biopsy of the allograft is not generally necessary.

A major potential complication in the immediate posttransplant period is infection. The only significant infections we have observed were in SC and AR who developed cytomegalovirus hepatitis and herpes simplex of the eye, respectively. In both instances, the infection was relatively mild.

We have observed several other problems in the posttransplant course of our patients: various degrees of hypertension, growth failure, and steroid-induced cataracts. Moreover, azathioprine has been associated with malignancy, but the follow-up of our patients has not been long enough to observe this potential complication.

(Continued on page 592)



The Ethical Context

Prenatal Diagnosis of Cystic Fibrosis and Other Genetic Diseases

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ADVANCEMENTS in every field of medicine have brought about increasing longevity and in turn have altered the spectrum of disease. The former roles of infectious diseases as the limiting factors in infant survival and as important determinants of survival through middle age have diminished in the light of sophisticated antimicrobial therapy. Pneumonia, while still a "friend of old age," is now particularly the friend of the elderly patient with severe chronic disease. As people live longer, protected from untimely death at the hands of contagion, they are afflicted by today's predominant diseases of old age, such as arteriosclerosis and cancer, and frequently succumb to infectious complications.

Survival to a productive older age is considered to make acceptable the increased incidence of the diseases of the elderly. The social costs of maintaining the health of a booming elderly populace are occasionally questioned in these days of financial preoccupation, but senior citizens can in no direct way be regarded as a threat to the vitality of the human race. On the other hand, improved therapeutics have also increased the longevity and the reproductive potential of another segment of the population, those born with birth defects.

Editor's Note: Papers published in the Student Thesis series have been written by fourth year medical students at the University of Kansas School of Medicine. Those selected for publication have been recommended by the UKSM faculty.

Popular from the time of Darwin into the middle of the twentieth century, eugenics lost much of its palatability through its grossly distorted practice in Nazi Germany. With its persistant negative connotations and its past abuse, the practice of eugenics remains unpopular when considered on its own merits. For this reason, human potential and social costs become major considerations, with the gene pool as a possible beneficiary. In diseases where mental retardation and physical deformity are severe, or when the quality of life is poor, decisions in favor of some form of intervention are frequently made. Considerations of financial and emotional burdens faced by a family or by society create ethical questions more difficult to resolve.

Expansion of the frontiers of medical science tends also to broaden the scope of ethical dilemma. Examined here are some moral, philosophic, and economic considerations when a genetic defect, but not its scope of ramifications, can be determined in utero.

The quality of the gene pool is of basic importance to those interested in eugenics. As the concept of selecting breeding is unacceptable in our society, the best means for improving the genetic make-up of the

population is through genetic counseling. By the avoidance of conception, a couple would be assured that their known status as carriers of a genetic disease would not be manifested as the disease itself in their offspring. Or, if conception occurs and the disease in question can be diagnosed prenatally, then selective abortion becomes an option.

Genetic defects are best divided into chromosomal disorders, sex-linked diseases, inborn errors of metabolism, and congenital malformations. Down's syndrome is a chromosomal disorder, involving an additional 21st chromosome. Prominent abnormalities in facial structures are obvious from birth; moderate to severe mental retardation is present. Diagnostic and surgical advancements have allowed the survival past adolescence of many of these children born with associated cardiac and gastrointestinal defects that would have otherwise been life-threatening. Without complicating illness or after successful treatment, the quality of an affected child's life could be considered acceptable; the impact that the Down's child has on its family is frequently less than positive, however, and the financial burden can be great.

Sex-linked diseases include hemophilia and Duchenne's muscular dystrophy. Males who inherit these diseases from their carrier mothers will be variably affected. Replacement of deficient clotting factors are an expensively effective treatment for the hemophiliac, who will nonetheless have periods of considerable disability. The boy with muscular dystrophy faces progressive weakness and, eventually, a premature death.

Inborn errors of metabolism have a wide variety of effects. Cystic fibrosis, dealt with in depth later, has a prolonged, progressive course which has been successfully extended by improved therapeutics in the absence of a known specific defect. Tay-Sachs disease, with a defined defect of lipid metabolism, causes severe, progressive psychomotor retardation and death in infancy or early childhood. Taken as a whole, the inherited biochemical disorders are estimated to occur at a frequency of up to 1 per cent of live births. While many of these disorders are minor and may never be diagnosed, the overall impact of this group of diseases is obviously immense.

The last group of hereditary defects are the congenital malformations. These, too, range in severity, from cosmetic defects with minimal disability (polydactyly), through malformations with both cosmetic and dysfunctional effects (cleft lip and palate), to severely disabling or fatal conditions such as neural tube defects.

With such disparity in the genetic diseases, a wide variety of methods for diagnosis, intervention, and treatment have been developed.

Structural abnormalities show up at birth; surface deformities are seen on initial inspection and severe organ system malformations typically reveal their presence within the first few days. Biochemical defects, on the other hand, may not create signs or symptoms for months or years. In any case, early recognition of defects often improves the prognosis. The mental retardation of a Down's child can only be modified, but associated heart or gastrointestinal anomalies, when corrected, can improve dramatically the patient's well-being. The mental retardation of phenylketonuria can be avoided if the defect is discovered by screening at birth and a restrictive diet is instituted.

There have thus evolved a number of methods by which defects can be recognized early. The simplest involves an association approach, that is, the suspicion of defects known to be associated with a recognizable syndrome. A baby born with facial features suggestive of Down's syndrome would have more attention paid to its vomiting or its heart murmur, given the higher incidence of gastrointestinal and heart defects in the syndrome.

More control can be achieved when screening for a disease is possible. A group known to be at high risk for a genetic disease, such as blacks for sickle cell anemia, would be subject to screening on a moderate scale. Large scale screening could disregard differences in risk, if any, and screen the entire population. An example of this sort of effort is the screening of newborns for phenylketonuria. A small scale screen is limited to children of families known to transmit a heritable disorder. Such screens can be exotic and expensive, but large scale operations require reliable, easily performed procedures that can be done economically and can hope to provide a cost-effective yield.

Early detection offers only the option of intervention in most genetic disorders. Intervention in this context requires contraception or abortion.

Autosomal recessive diseases are inherited from both parents, who must each carry the trait as heterozygotes or be homozygous and have the disease. Preventive contraception can be advised when it is possible to identify both parents as carriers of a certain disease trait. Identification can be based on a history of a previous child having been born with the disease or, in some cases, by laboratory screening tests. Tay-Sachs disease, sickle cell anemia and, probably, cystic fibrosis are examples of diseases for

which the heterozygotic state can be detected by screening. As in screening for infants born with genetic diseases, the laboratory detection of carriers can occur at several levels. Most techniques are sufficiently specialized to restrict their use to high risk individuals or groups. Programs have thus been instituted to screen blacks for sickle cell trait and Ashkenazi Jews for Tay-Sachs disease trait.

Amniocentesis has permitted the prenatal diagnoses of many genetic disorders. Its earliest application was in the detection of chromosomal abnormalities. Down's syndrome, with its extra 21st chromosome, could be readily detected. In sex-linked disorders, a male fetus with a carrier mother has a 50 per cent chance of having the disease. By shedding into his surrounding amniotic fluid cells with the male karyotype, the male is identified; one half of the male fetuses thus detected would actually be free from the disease.

Tissue cultures of fibroblasts obtained by amniocentesis can be subjected to a wide range of biochemical studies. In this manner, more than 100 inborn errors of metabolism can be detected before birth. Most of these diseases are so rare that routine efforts to screen amniotic fluid samples for their markers is out of the question. Further, tissue culture is a difficult procedure requiring several weeks to provide enough material for such assays; any loss of a sample would cause a delay in the diagnosis and eliminate selective abortion as an option.

The most practical assays are run directly on fresh amniotic fluid. Available now are tests for fetal lung maturity and for neural tube defects. The latter involves an assay of amniotic alpha-fetoprotein, and is in surprisingly wide use as an indication for selective abortion. Any practical assay to be used on a large scale will have to be of this type in order to provide the necessary reliability at a reasonable cost.

Cystic Fibrosis

Cystic fibrosis is the most common severe genetically transmitted disease in the United States. As a result, there is a substantial population of chronically ill patients afflicted with the disease, with all of the attendant suffering, family stress, and economic burden. A disease with such an impact will always generate research efforts directed toward its treatment, its cause, and its prevention. In the case of genetic disease, prevention involves a variety of ethical issues; these will be explored as they pertain to cystic fibrosis in a later section.

Cystic fibrosis (CF) is transmitted as an autosomal recessive trait. In order to be expressed, the trait must have been inherited from both parents. Such a union would result in the classic ratio of 1:2:1 for noncarriers, carriers, and affected offspring. This inheritance pattern in a disease usually suggests a specific defect in a biochemical pathway or cellular structure. This defect has been difficult to detect in CF, leading some investigators to suggest that multiple alleles may cause different defects leading to the same disease complex. The best studies have concluded that two or three loci are involved, but even these results have not been easily reproducible.

The significance of cystic fibrosis lies in the high frequency with which the gene (or genes) responsible for it is carried in certain populations. Based on an incidence of the disease of 1/2500 to 1/2000 among Americans of northern European descent, the carrier rate for that population is in the range of 4-5 per cent; the incidence is much lower in blacks, southern Europeans, Asians, and Orientals. A carrier rate of 4 per cent or greater in an easily defined population is usually considered sufficient to allow practical application of screening measures. This potential in cystic fibrosis will be discussed below.

The diagnosis of cystic fibrosis is rarely made without a positive sweat test (measured as increased chloride concentration in the sweat). Usually at least one of three other criteria must also be present. These include chronic obstructive pulmonary disease, exocrine pancreatic insufficiency, and a family history of CF. The first of these is almost always present, while the second is less universal.

The patient may present in the neonatal period with symptoms of meconium ileus. Older children might present with respiratory symptoms. A cough that begins nonproductively usually progresses to a productive one, accompanied by decreased exercise tolerance and increased respiratory rate. Pancreatic insufficiency occurs in 80-90 per cent of patients with CF.[1] This is manifested by symptoms of maldigestion, such as steatorrhea and excessive flatus, accompanied by poor growth and delayed maturation.

Less obvious problems occur in the liver and biliary systems. Fatty change in the liver is relatively common, and occurs even with adequate diet and pancreatic enzyme replacement. Focal biliary cirrhosis, in a form unique to CF, is found on autopsy in 20-25 per cent of patients.[1] It becomes clinically apparent in older patients, and has increased in importance as life expectancy has improved.

Infertility in postpubertal males with CF is estimated to be around 98 per cent.[1] It is thought to be due either to developmental failure or to intrauterine

obstruction of the vas deferens and seminal vesicles, which will be absent or show atrophy. In contrast, women CF patients do not have reproductive abnormalities, although secondary amenorrhea due to malnutrition or severe pulmonary disease may occur. Also, changes in cervical secretions occur, which may or may not affect fertility.

Complications are the rule in cystic fibrosis; their prevention and treatment have contributed to an increased life expectancy for those with the disease. *Pseudomonas aeruginosa* has replaced *Staphylococcus aureus* as the predominant isolate from the pulmonary secretions of CF patients. Such chronically present bacterial infections are exacerbated by viral infections and frequently lead to pneumonia.

Progressive airway obstruction may lead to bronchiectasis and a worsened prognosis. Other pulmonary complications include pneumothorax, atelectasis, hemoptysis, and cor pulmonale. Intestinal complications include obstruction and intussusception. Complications of cirrhosis will occur, including portal hypertension and hepatic failure, with their multisystem effects.

In general, the life expectancy of cystic fibrosis patients has been found to be greater when the disease is diagnosed prior to the onset of pulmonary disease. Scored chest x-rays and lung function tests, when measured in 7-year-old CF patients paired with their affected siblings, were significantly better in the younger siblings, all of whom had been diagnosed before the age of 1 year.[2] This group scored better on most parameters, and was found to display less general morbidity than their older siblings at the same age. While these results are impressive, it must be noted that other studies have failed to confirm these findings.[3]

When diagnosis and treatment have been possible before pulmonary involvement became irreversible, the survival rate for cystic fibrosis patients has exceeded 96 per cent following 18 years of treatment.[1] The national survival rate was 50 per cent at 16 years of age in 1975, with a projected rate of 50 per cent at 19 years of age in 1980.[3] Comprehensive care in regional centers in Australia has resulted in a survival rate of 50 per cent at 26 years of age.[4]

So, in terms of actual survival, the prognosis for CF patients is improving. Treatment of vitamin deficiency, pancreatin administration to overcome pancreatic insufficiency, advances in antimicrobial drugs, earlier and more accurate diagnosis, intensive respiratory therapy, and prompt intervention in the face of complications have all contributed to increasing the life expectancy of these patients.

Obviously, the number of patients with treated CF and their average age are both increasing. Their quality of life, especially when compared to that experienced years ago, is also improving. Many patients function well in society's mainstream, with some limitations.

Optimism in the face of this improving picture must be opposed by realistic observations that first, cystic fibrosis patients are not cured, and also, that mean life expectancies in the 20-30 year range would not be acceptable to the general population. CF patients are chronically ill, and can be expected eventually to succumb to their disease or its complications.

Relatively good years experienced by the well treated patient are made possible by medications and periodic hospitalizations. Respiratory infections will occur even with the most comprehensive maintenance, and often will require sophisticated antibiotics to resolve. Increased pulmonary involvement will lead to pulmonary hypertension with increasing age, requiring intensive treatment to delay the eventual onset of cor pulmonale. When this stage is reached, the best treatment can maintain a patient for several years, but not without discomfort and an ever increasing financial burden.

Cystic fibrosis is a mechanistically complicated disease. A wide variety of defects have been discovered in CF patients, but the issue of cause *vs* effect has yet to be satisfactorily answered for many of these. Conflicting results have confused the issue, but certain concepts are becoming increasingly more credible.

The major defect in CF is considered to be exocrine gland dysfunction. This occurs at a multitude of sites, including lung airways, salivary glands, small intestine, pancreas, biliary system, cervix, and male genital tract. Decreased water content, altered electrolyte concentrations, and a change in the composition of organic substances have all been proposed as contributory to subsequent obstruction.

Other systems found to be dysfunctional include cellular membranes, local immune responses, and the mucociliary transport system. The nature of the defect in membrane transport has been difficult to determine, but it is generally thought to be secondary to the unknown primary abnormality; it results in the elevated sodium and chloride concentrations in patients' sweat and other secretions. Locally diminished immune responses are suggested by the rarity of systemic infections in the presence of chronic pulmonary infection. Mucociliary transport, which normally prevents the accumulation of inhaled particles and natural material in the pulmonary passages, is considered to be impaired, but not held

to be a primary defect.

A "cystic fibrosis protein" (CFP) has been discovered in the serum of both CF patients and carriers of the trait. A plausible disease mechanism assigns the primary defect to an abnormal alpha-2 macroglobulin.[5] Its altered regulatory role on the activity of various proteases is postulated to result in increased levels of CFP and other biologically active substances in the serum. These substances are held to directly alter ion transport across membranes, leading to the abnormal composition of secretory fluids. This combined with altered ciliary transport is believed to be the cause of organ obstruction.

Inborn errors of metabolism are manifested in patients by deficiencies of normal substances, the presence of abnormal substances, or by the accumulation of waste or intermediate products that would be further processed by a normal system. In the hypothetical CFP model presented above, the defect results in the accumulation of CFP. The model is hypothetical in that the role of the alpha-2 macroglobulin remains in question, and the biological role of CFP can only be postulated. But the accumulation of CFP to detectable levels in CF patients and carriers has been adequately documented, as has the abnormal activity of certain proteases.

CFP assays require carefully prepared serum and a complicated technique called isoelectric focusing.[6] When precise guidelines have been followed for serum sample processing, the assay has shown significant accuracy.[5, 7] This assay does have deficiencies beyond its difficult design. While it will detect CF carriers, its qualitative nature prevents differentiating them from CF patients. This would make it ineffective for neonatal screening. Further, the detection of a small but significant portion of CF patients who are CFP-negative suggests a distinct phenotype for this subgroup, and creates additional limitations of this assay.

A refinement on the CFP assay has been developed. Pooled antisera from animals injected with isolated human CFP is absorbed with CFP-negative human sera to yield a CFP-reactive antiserum. Used as an immunoassay for detecting CFP in test sera, this "anti-CFP" has duplicated the accuracy of the complicated CFP assays. But more importantly, it is sufficiently quantitative to differentiate heterozygotes or carriers from CF patients.[8] If it were possible to produce the CFP-antiserum in sufficient quantity, mass screening would become a practical possibility.

In fact, more recent work suggests that the potential for this assay is great. A "hybridoma" cell line has been developed which combines an easily cultured mouse myeloma cell strain with murine spleen cells that have been made immune to CFP. This tissue culture successfully produces mouse antibody to CFP.[9] Isolation of a cell line specific for anti-CFP should be possible, thus providing an inexpensive source of large amounts of the antiserum. Screening by this method would be fast, simple, and reliable; the false negative rate for CFP assays is less than 10 per cent.[8]

It must be noted that the prenatal diagnostic potential for CFP assays has not been explored.

It has been mentioned that the speculative and defective alpha-2 macroglobulin is expected to play its role in the abnormal regulation of certain proteases. Of special interest is arginine esterase. The activity of this enzyme can be determined by titration with the compound 4-methylumbelliferyl-guanidinobenzoate (MUGB). In some careful studies, arginine esterase activity has been shown to be reduced in CF patients, carriers, and in the amniotic fluid of babies who when born were found to have cystic fibrosis. Three hundred second trimester amniotic fluid samples monitored for reduced arginine esterase activity correctly predicted the three infants who would subsequently be affected by CF.[10] Further refinements in technique by the same study group allowed correct prediction of outcome of 13 consecutive pregnancies known to be at risk.[11]

A technique based on a presumably different primary defect, which has not been speculated upon, bears discussion. It has been proposed that the defect causes an abnormal recognition of hydrolytic enzymes by cell membranes. The enzymes are rendered thermolabile in the presence of otherwise normal activity. It has been shown that control plasma contains an alpha-mannosidase that is heat stabile, while CF carriers and patients contain in their plasma alpha-mannosidase enzymes that are progressively more heat labile.[12] The results appear quite conclusive but, as in the case of CFP assays, no role is proposed for these enzyme thermolability studies in the prenatal diagnosis of cystic fibrosis.

Notable also is a recent report that cultured fibroblasts from CF patients accumulate less sodium under controlled conditions than do normal cells. By using the sodium-22 isotope, significant differences in accumulation allow the differentiation of not only CF patients from normal individuals, but also the identification of obligate carriers.[13] This is another example of a cumbersome, awkward test with minimal applicability for widespread screening. It does, however, have important differences from the others that have been mentioned. It is available immediately and has been shown to be very accurate.

And, for those who consider transport alterations to be the primary CF defect, this method may be more acceptable; it can be regarded in this context as being "closer to the gene."[14]

The practical use of the sodium transport assay will be limited to the screening of high risk individuals, such as parents who fear that they may be carriers, and children of obligate heterozygotes. Its use in prenatal diagnosis can be considered. Fibroblasts cultured from patients with other inborn metabolic defects, such as Tay-Sachs disease, typically reveal biochemical defects in common with those of cultured cells obtained by amniocentesis. This technique could probably be applied to diagnose fetuses affected by CF but, as has been previously mentioned, a direct assay will always be superior to one that involves the tenuous culture of amniotic fluid cells.

Diagnosis of cystic fibrosis can be made at all levels. At the extreme, it can be suspected in an adult with unexplained portal hypertension, steatorrhea or obstructive pulmonary disease, and diagnosed by the conventional sweat chloride test. A child with pulmonary disease or delayed growth, or an infant with meconium ileus can be diagnosed in the same manner.

At the next level, diagnosis is now possible before the appearance of pulmonary disease by the measurement of sodium transport in fibroblast culture in children at high risk. In the future, anti-CFP immunoassay should make such screening available routinely in pediatric practice. An earlier level of detection using a simple method such as the immunoassay may become practical, allowing routine premarital screening to detect carriers. The presently available cell culture method could be used only to screen couples at high risk. Two carriers who might choose to have children anyway would have available to them amniocentesis and selective abortion.

The technique with the greatest potential for large scale screening appears to be that using CFP antiserum. A large prospective study employing anti-CFP will prove or disprove its efficacy. Less attractive for screening but more so for prenatal diagnosis is the MUGB assay for arginine esterase activity. The other methods presented are all thought to be too complicated in their present states for employment much beyond the experimental stage.

Ethical Considerations

We have seen the potential of diagnostic genetics. Amniocentesis is presently employed to prevent the birth of defective infants, and its use may rapidly expand to many more applications, specifically in providing for selective abortion for a lengthening list of diseases.

The special situation of cystic fibrosis was selected for a number of reasons. Technologically, the methods are interesting and present well the many facets of the disease and its diagnosis. But the emotional issues are also of interest in this disease where the decisions are not so clear cut as in some other birth defects. Cystic fibrosis is a serious disease and carries a grave ultimate prognosis, but the quality of life in the interim can be very good. And now genetic technology, in its expanding sphere of applications, may soon make the birth of a child with CF an avoidable event.

The abortion issue itself must be superficially considered in a discussion centered on selective abortion. Some people approach all morality questions on an all-or-nothing, absolute right/absolute wrong basis. Such conservativism lends itself more frequently to the anti-abortion individual. Abortion can be taken to be wrong in general and not allowable under any circumstances. With this orientation, it is held that there are definite answers for every issue, and that situational accommodations are immoral when they stray from an ordained set of answers.

To this group, the question of selective abortion need not be directed. They can be comfortable in their consistency, and untroubled by the struggle to distinguish different shades in the gray area of this issue.

On the other hand, perhaps the majority of people would allow abortion under certain circumstances. Often cited as acceptable, even to those generally against abortion, is the true therapeutic abortion undertaken to save the mother's life. Acceptable to a lesser degree is abortion in cases of rape and incest. Least acceptable are abortions performed for the convenience of the mother, although many would allow considerable latitude for socioeconomic factors or even for a woman's right to choose.

Genetic diseases present themselves in every degree of severity. Different scales might be arranged in terms of quality of life, physical discomfort, social and financial costs, and so on. It cannot be expected that society will ever accept selective abortion for every genetic disease. Distinction of the position of a disease on some "master" scale might ease the decision if it were possible, but there would still be no allowance for individual variance.

Mothers at risk are presently screened for Trisomy-21, or Down's syndrome. Parents who have effectively dealt with the problems of raising an affected child speak frequently with great conviction

about the positive effect the experience has had on their families. In addition, mental retardation, physical disability, and life expectancy all show considerable variability in this disease. Even so, screening with resultant selective abortion has been going on for years and has received surprisingly little attention. This common, usually severe syndrome is accepted as warranting a degree of intervention.

An easier choice might be made in the case of neural tube defects, readily predictable by measuring amniotic fluid levels of alpha-fetoprotein. Anencephaly is a horrible defect; few could support bringing an affected fetus to term. Myelomeningocele complicates a clear-cut anencephaly decision by presenting with the same result on amniocentesis assay. Although highly variable in its severity, myelomeningocele is typically profoundly disabling and requires complicated surgery to allow a life of disputable quality. Like Down's syndrome, neural tube defects are screened for, and affected fetuses are frequently aborted. A choice is being made by parents who have hopefully been well counseled; justification is apparently found to favor abortion in this disease.

More controversial is abortion of male fetuses known to have a 50 per cent risk of having a sex-linked disorder such as Duchenne's muscular dystrophy. There obviously exists a 50 per cent chance that a normal male fetus will be aborted. In spite of this discouraging yield, such screening is practiced, allowing a genetically counseled couple an alternative to avoiding future pregnancies.

The prenatal diagnosis of cystic fibrosis is an issue not easily resolved. CF patients achieve all levels of education, occupy all kinds of jobs and professions, and may spend their shortened lives in relative freedom from disability. Those with the disease who do not reach the average level of accomplishment or the average life span may also have many good years when their disease is in fairly good control. The extremely ill child with complicated disease, severe discomfort, and death in early childhood is now the exception.

There are factors that negatively influence this situation. As has been discussed, there are exacerbations of the disease. Intensive and expensive therapeutic interventions are required. Eventual development of pulmonary pathology will disable even the most productive patient, and death from cardiopulmonary failure will ultimately follow.

Cystic fibrosis is uncurable. It is preventable now by one method only — the avoidance of conception. In five to ten years, a second method will probably be considered reliable enough for use in at least limited situations. Prenatal diagnosis of CF seems to be inevitable.

Selective abortion for CF would be completely justifiable to one who holds eugenic views. The high incidence of the trait and the disease coupled with prolonged survival of CF patients make this disease detrimental to the overall genetic quality of the population. A significant drain is applied to the gene pool by the presence of this disease with its constant 5 per cent carrier rate, forever supplying the population with afflicted individuals.

Even this hard eugenic line makes no mention of condemning those living with the disease. The "slippery slope" argument that suggests that infanticide will develop by simple progression cannot be valid. "Slope" arguments are applied to passive euthanasia, geriatric medicine, scientific research, social affairs, and all sorts of other things. The avoidance of any form of manipulation or intervention to prevent progression down the slope to chaos and immorality also precludes advancement in any of these areas.

Advocates of selective abortion consider themselves to be taking an active role in the prevention of the birth of affected children. They are comfortable with the difference between a 15-week-old fetus and a new born infant, and feel no compulsion to slide down the slope toward infanticide. Those who do not differentiate abortion at any stage from infanticide would shrug off the distinction, but for them the slope does not exist and the issue has been long closed anyway.

Eugenists see their views as humanistic. They have the population's good as their goal. A more conventional humanist, or a situational ethicist, steps around the unpopular notion of eugenics, avoiding arbitrary judgments of what is genetically good by tending to evaluations of the "overall" good. S/he would prevent the birth of afflicted children to alleviate the suffering caused by the disease, rather than to improve the gene pool. The afflicted infant would not be born to suffer illness, its family would not have to cope with emotional and financial hardships, and society could escape from additional stress on its medical and support structures. A humanistic approach presumably weighs more factors than a eugenic approach, and might be hoped to attain balance by a cancellation of opposing errors.

There are several negative aspects to the issue of selectively aborting fetuses identified as having cystic fibrosis. For one, it could never be expected that the screening process would pick up all affected fetuses. What had been an unavoidable, not so uncommon disease would become not only avoidable

but also rare. The afflicted child would be a rarity, and although s/he would likely be well-treated medically, society's approach might be discriminatory.

It is obvious that no diagnostic procedure will be able to identify the exceptional CF-afflicted fetus, the one with minimal disability from the disease and destined for prolonged survival. The loss of fetuses with such potential weighs negatively against a comprehensive prenatal screening program for CF.

Practical consideration of this issue yields no simple answers. If the decision could be made without requiring abortion to carry out the choice, no parents would ever choose to have a child with cystic fibrosis. In fact, it seems that parents not willing to tangle with the ethical questions of selective abortion may have an obligation to not bear children at all. Avoidance of pregnancy does afford a completely effective preventive measure for couples known to be at risk, and may represent a truly responsible decision.

One in four children born to parents carrying the trait for cystic fibrosis will have the disease. To knowingly allow a pregnancy to begin in light of this risk seems irresponsible; by some standards, it could be considered immoral.

Decisions to prevent cystic fibrosis might be assigned a comparative morality rating as a component of the "master scale" discussed earlier. Avoiding pregnancy would rate very high, especially since it also avoids the abortion issues. The lowest rating would go to a high risk pregnancy allowed to proceed without consideration of prenatal diagnosis. Selective abortion would achieve an intermediate rating — high or low depending upon how abortion in general is viewed.

Other components are included in the humanistic approach. This would accommodate a person's right to become a parent and society's rights to limit population and increase its general health. Selective abortion gains acceptability as a moral decision under such situational ethics, while the risk-taking pregnancy continues to be the worst possible choice.

In present use, amniocentesis is typically monitored for genetic disease only in high risk groups or when a sample is obtained for other purposes. An exception is alpha-fetoprotein monitoring in Great Britain, where serum levels of the marker are routinely obtained. An elevated level in the maternal serum correlates well enough with fetal neural tube disease that it is considered an indication for diagnostic amniocentesis. Serum alpha-fetoprotein assays will soon be available in the United States, and routine screening may occur for neural tube diseases here once FDA approval is obtained.[15]

Cystic fibrosis, as an autosomal recessive disease, allows screening by carrier detection. Reliable techniques will soon provide adequate identification of carriers, and could conceivably be applied on a large scale. Certainly, historical prediction or laboratory identification of a carrier would be an indication for screening of an intended spouse for the trait as well.

It cannot be expected that CF detection will proceed beyond such modest selective screening. It is interesting to note that cystic fibrosis is carried as a trait in the population of American whites at about the same rate as the sickle cell trait is carried by black Americans. The incidence of both diseases is also quite similar. Sickle cell trait screening is widely publicized and is performed on a large scale, with the intent to provide genetic counseling and thus decrease the number of children born with the disease.

Cystic fibrosis and sickle cell anemia both respond to careful medical management to provide those affected with a stable clinical course and the potential for a productive life. Both diseases allow similar, shortened life spans which have lengthened with therapeutic advances.

Some blacks have argued that sickle cell trait screening is a racist practice, perhaps inadvertently pushed by the white-dominated medical establishment to control the reproductive rights of black people. One can at best be skeptical about how enthusiastically that establishment, or the white American population itself, would embrace a screening program with the same theoretical yield for cystic fibrosis. It seems reasonable to predict that the potential for large scale CF screening will be greatest if the above inconsistency and comparison becomes general knowledge.

A brief discussion of this nature will never be sufficient to cover every angle of a complex ethical question. What has been done is to carefully present some of the issues with the attitude that for some things, there are no definite and absolute answers. At times, situational considerations are the most important.

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Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Ascites

A Diagnostic and Therapeutic Approach

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Ascites alerts both the patient and the physician to the presence of a serious health problem. Even early physicians were well aware of the grave prognosis associated with ascites. Hippocrates observed, "When the liver is full of fluid and this overflows into the peritoneal cavity so that the belly becomes full of water, death follows." Although modern medicine has provided more effective therapy for patients with cirrhosis who develop ascites, overall patient survival at two years is still only 40 per cent. Nevertheless, it is important to attempt control of ascites, as the condition may foster potentially lethal complications such as variceal hemorrhage, hepatorenal syndrome, and spontaneous bacterial peritonitis. Ascites also may cause anorexia or atelectasis by compression of contiguous organs, or may induce adbominal hernias. Pleural effusion, usually right-sided, accumulates because of a diaphragmatic defect in about 6 per cent of patients. Interestingly, pleural effusion may resolve only after ascites is eliminated completely.

Pathophysiology

Several factors must be present for ascites to develop in the patient with cirrhosis. First, Starling equilibrium forces are altered by diminished colloid osmotic pressure (hypoalbuminemia) and increased sinusoidal hydrostatic pressure (portal hypertension). Second, obstruction to portal blood flow must be intrahepatic (cirrhosis) or post-sinusoidal (Budd-Chiari syndrome), suggesting that altered hepatic lymphatic drainage is important. Third, sodium retention by the kidney plays an important role.

Two theories have been proposed to account for the excessive sodium retention associated with cirrhosis *(Figure 1)*. Proponents of the classic theory argue that altered Starling equilibrium forces cause

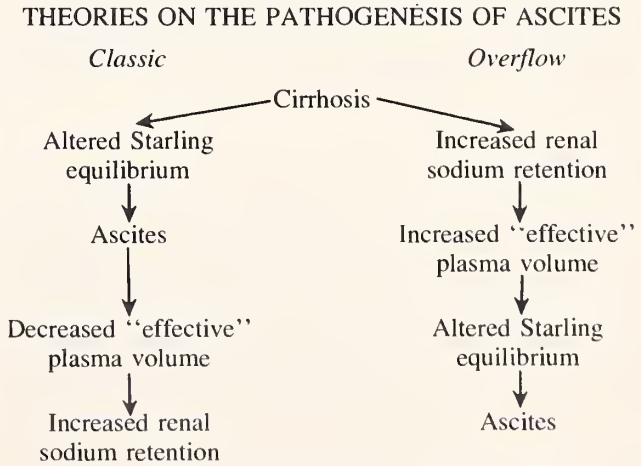


Figure 1. The pathogenesis of ascites in cirrhosis may be explained by two theories: the "classic" theory in which the primary defect is altered Starling equilibrium or the "overflow" theory in which the defect is an abnormal renal sodium retention.

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peritoneal transudation of fluid. Ascites accumulation causes a decrease in "effective" plasma volume which, in turn, stimulates renal sodium retention. In contrast, proponents of the overflow therapy argue that the primary problem is increased renal sodium retention, which causes ascites because of altered Starling forces. Central to both theories is the important clinical concept that patients with cirrhosis are unable to excrete large amounts of salt. Serial observations in dogs with cirrhosis induced by dimethylnitrosamine show that sodium retention occurs long before ascites develops, supporting the overflow theory.

The cause of enhanced sodium retention is not clear. It is probably due to a combination of factors including: hyperaldosteronism; altered intra-renal blood flow; renal prostaglandin abnormalities; increased sympathetic nervous system activity; kallikrein-kinin abnormalities; deficiency of a putative humoral naturetic factor; and hyperestrogenemia.

Diagnosis

When a patient with liver disease gains 20 pounds rapidly and has the triad of bulging flanks, shifting dullness and a fluid wave, the diagnosis of ascites is obvious and easy. Often, one can ballot the liver or spleen. In many situations, however, the patient complains of a slight weight gain and perhaps a feeling of fullness or an expanding waist line. In the patient with questionable ascites, careful examination by experienced clinicians has an accuracy of just over 50 per cent. Ultrasonography, which can detect as little as 100 ml of fluid, is recommended for definitive evaluation in such instances. Because increased sodium retention is a prerequisite for the development of ascites, a random urine specimen should have a low (< 20 mEq/L) sodium content if the patient is developing ascites and is not on diuretics.

Paracentesis

Once the presence of ascites is established, the underlying cause must be identified. Although 90 per cent or more of patients will have cirrhosis, neoplasm or cardiac disease, the differential diagnosis is broad *(Table I)*. Analysis of ascitic fluid usually proves helpful. A preferred site for paracentesis is the avascular midline, midway between the umbilicus and pubic symphysis. After the patient has voided, the site is prepared with aseptic technique and a small caliber needle (20G) is advanced through the anesthetized site until there is return of fluid. In patients with tense ascites, a Z-track or angled insertion technique may help occlude the needle track after paracentesis. A needle is never inserted through a surgical scar since bowel tissue tends to adhere to the anterior abdominal wall incision, increasing the risk of bowel or mesenteric vein puncture. If the midline cannot be used, the right or left lower quadrant is acceptable provided care is taken not to insert the needle through a visible venous collateral. Upper quadrants are avoided because of the possibility of hepatosplenomegaly.

It should be stressed that there are no absolute contraindications to a diagnostic paracentesis if the information is important for patient management, such as exclusion of bacterial peritonitis as a cause of clinical decompensation. Even if the patient has an associated coagulopathy, the diagnostic yield usually outweighs the risk of a carefully placed small gauge needle. Some authorities infuse fresh frozen plasma before paracentesis if the prothrombin time is prolonged more than three seconds. Since some patients have a thick panniculus, a dry tap may occur if the needle does not enter the peritoneal space. If the attempted paracentesis is unsuccessful using standard technique then ultrasound can help direct future attempts to obtain fluid.

TABLE I
DIFFERENTIAL DIAGNOSIS OF ASCITES

Condition	*Usual Causes*
A. No peritoneal disease	
1. Portal hypertension	Cirrhosis; heart failure or constrictive pericarditis; portal vein, hepatic vein or inferior vena cava thrombosis
2. Hypoalbuminemia	Kwashiorkor; nephrotic syndrome
3. Miscellaneous	Ovarian disease; myxedema; pancreatic, biliary, or ureteral fistula; chylous or nephrogenic (hemodialysis) ascites; decapsulation syndrome after renal transplantation
B. Peritoneal disease	
1. Infection	Spontaneous or nonspontaneous bacterial peritonitis; tuberculosis, fungal or parasitic infections
2. Neoplasm	Ovarian, hepatoma
3. Miscellaneous	Eosinophilic gastroenteritis; Whipple's disease; vasculitis; granulomatous disease
C. Pseudo-ascites	Obesity; pancreatic pseudocyst; hydronephrosis or urinary retention; ovarian or mesenteric cyst

Ascitic Fluid Analysis

Ascitic fluid usually appears straw-colored or perhaps weakly bile tinged. Deep bile staining suggests a biliary tract-peritoneal fistula, which is usually associated with trauma, biliary stones, or carcinoma. A turbid, malodorous fluid suggests infection. Chronic chylous (milky) ascites occurs with lymphatic obstruction; more than 80 per cent of the time it is associated with a neoplasm, usually lymphoma. Chylous fluid layers on standing, stains positively for fat, and triglyceride levels exceed those in the plasma. The latter is important since the opalescence of pseudochylous ascites results from phospholipid-protein material released by degenerating cells, and triglycerides are not elevated. Hemorrhagic ascites (RBC > 50,000/cu mm) is usually associated with ischemic necrosis or exfoliation of a primary liver or ovarian carcinoma.

It has long been thought that ascitic fluid can be separated into two categories on the basis of chemical analysis: transudate (specific gravity < 1,016, protein < 3 g/dl) or exudate. It is now recognized that this distinction is of limited value because of wide overlap between values. Long standing transudates tend to develop exudative-like characteristics, and diuresis causes cell-free reabsorption of fluid, which increases the total cell count and protein level. Nevertheless, an extremely high protein concentration might suggest malignancy, myxedema, or tuberculosis. Fluid from diagnostic taps should always be analyzed for total cell count with differential, culture (aerobic and anaerobic), and amylase.

Spontaneous Bacterial Peritonitis

This serious complication in patients with ascites may appear in the absence of an obvious precipitating event. Bacteremia with contamination of ascites is postulated as the most likely cause. A single organism, usually an enteric pathogen, but less commonly a streptococcus, is responsible in 90 per cent of cases. Although rarely the patient may have no signs or symptoms, there is usually associated fever, abdominal pain, portal-systemic encephalopathy, or peripheral leukocytosis. An ascitic fluid absolute polymorphonuclear leukocyte count greater than 500/cu mm or the presence of >500 polys/cu mm but signs of peritonitis is highly suggestive of spontaneous bacterial peritonitis. These patients should be started on broad spectrum antibiotics until cultures and sensitivities are available. Patients with an ascitic fluid count less than 500 polymorphonuclear leukocytes, but no signs or symptoms suggestive of bacterial peritonitis, are not treated empirically. Recent observations indicate that the ascitic fluid pH in patients with alcoholic cirrhosis and spontaneous bacterial peritonitis is markedly diminished (mean 7.25 ± 0.06) while the mean blood pH is normal. In contrast, patients with sterile ascites had a mean pH of 7.47 ± 0.07. Thus, ascitic fluid pH may become an important determinant in deciding if spontaneous bacterial peritonitis is present before culture results are available. If the syringe is tightly capped and kept on ice, samples for pH determination remain reliable for approximately 30 minutes. One caveat worth remembering is that patients with systemic acidosis and sterile ascites, as may occur in renal failure, will also have a low ascitic fluid pH.

Nonspontaneous bacterial peritonitis, which is usually associated with a perforated ulcer, appendix or colonic diverticulum, occurs in 10 per cent of patients with ascites and peritonitis. Findings characteristic of this entity are free gas on abdominal films and putrid ascitic fluid with leukocytes > 10,000/cu mm. Culture usually demonstrates multiple organisms including anaerobes.

Pancreatic Ascites

An ascitic fluid amylase greater than the serum value suggests a diagnosis of pancreatic ascites, a condition in which there is a fistula between the pancreatic duct and the peritoneal cavity. Pancreatic ascites can be missed if this test is not performed. Treatment may require surgical correction of the underlying pseudocyst or fistula.

Miscellaneous

In rare cases, the ascitic fluid may have marked eosinophilia, which suggests the diagnosis of hypereosinophilic syndrome or eosinophilic gastroenteritis. Ascitic fluid in tuberculous peritonitis often has a predominant monocytosis but is hemorrhagic in < 3 per cent of cases. The AFB stain is characteristically negative and culture provides a low yield. Therefore the diagnosis is best established by laparoscopic-directed peritoneal biopsy.

Although cytology is usually obtained, some authorities believe that routine cytology is unproductive since atypical mesothelial cells may resemble malignant cells, leading to a high rate of false positives. Others disagree, arguing that a false positive Class V cytology is rare. If malignancy is a diagnostic consideration, a volume of fluid sufficient for a cell block (100 ml or greater) should be sent for examination. Malignant ascites can be diagnosed with an overall accuracy of 60-90 per cent.

TABLE II
ABCs OF SODIUM BALANCE

A. A molecule of salt (NaCl) is 39% sodium by weight
Therefore, 5 grams NaCl contains: 39% × 5g, or 2 grams Na
B. The molecular weight of sodium is 23

$$\frac{2000\text{mg Na}}{23} = 87\text{mEq Na}$$

C. 140mEq Na loss obligates 1 liter water loss or 2.2 lb
D. A teaspoon of salt is 2400mg or 104mEq Na
E. Sodium restriction:

Mild	2400mg	100mEq
Moderate	1000mg	43mEq
Strict	500mg	22mEq

Treatment

For ascites due to cirrhosis, sodium restriction is the cornerstone of therapy. Most authorities recommend a diet limited to 20-40 mEq (500-1000 mg) of sodium daily. Since patients lose about 20mEq sodium per day by extrarenal means, a 20mEq diet will prevent weight gain and effect weight loss if any sodium is excreted in the urine. Some authorities would argue that water restriction is unnecessary unless the patient develops spontaneous hyponatremia. From a practical standpoint, however, it seems reasonable to limit the daily fluid intake to 1500 ml initially and to reduce this amount should hyponatremia occur. A recent survey of patients hospitalized in a regional hospital in Spain showed that spontaneous diuresis occurred in 28 per cent with bedrest and sodium restriction alone. A 24-hour urine collection to assess sodium excretion is important for both treatment and prognosis. One study of nonazotemic cirrhotics with ascites demonstrated that those with a 24-hour urine sodium excretion of less than 10mEq had a mean survival of six months, as opposed to 28 months in those patients with higher sodium excretion. The group with low urine sodium excretion also had elevated plasma renin, aldosterone and wedged hepatic venous pressures, but urine sodium was as discriminatory as the more sophisticated tests.

Calculations using the prescribed sodium intake, the 24-hour urine sodium excretion, and daily body weight can help identify the existence of unsuspected salt intake *(Table II)*. For example, a patient on a 40mEq diet who excretes 120mEq of sodium daily in the urine and has no weight loss, or perhaps weight gain, must have undetected salt intake. Often the source is antacids, medications, or surreptitious addition of salt to the unpalatable low sodium diet, either by a purloined salt shaker or high salt snacks brought to the hospital by friends. At home, patients may use baking soda ($NaHCO_3$) in large quantities for indigestion.

Previously, it appeared that patients treated with diuretics had an increased frequency of complications in comparison with those treated with fluid and salt restriction alone. A recent, controlled trial from Stanford University discounts this idea and shows that diuresis can be accomplished in seriously ill patients without complications attributable to diuretic therapy alone. Unless the patient has a markedly elevated urine sodium, early initiation of diuretic therapy is appropriate if there is no evidence of renal compromise. Spironolactone is the choice of most clinicians as the initial diuretic. Several studies have demonstrated that up to 75 per cent of patients respond to spironolactone alone without other diuretic intervention. Therapy should begin with 100-200 mg daily administered in a divided dose (since the drug half-life is 16 hours, twice daily is reasonable). Because spironolactone takes several days to achieve maximum effect, the dosage should not be increased unless there is no evidence of weight loss or diuresis after four days. An effective dose of spironolactone will cause a reversal of the urine sodium and potassium ratio; once the urine sodium is greater than potassium, diuresis should ensue. Most experts limit the daily dose of spironolactone to 400 mg. If diuresis does not occur, 40 mg of furosemide increased in a stepwise fashion every two to three days to a maximum dose of 120 mg/day should be added. Spironolactone therapy can cause hyperkalemia, usually precipitated by concurrent use of salt substitutes (KCl), or hyperchloremic acidosis related to iatrogenic hypoaldosteronism. Painful gynecomastia, an antiandrogen effect, sometimes limits spironolactone use in men.

Ascitic fluid is secreted by the visceral peritoneum and resorbed by the parietal peritoneum. Elegant studies have demonstrated that despite rapid diuresis, the maximum rate of ascitic fluid resorp-

TABLE III
CRITERIA FOR DIAGNOSIS OF REFRACTORY ASCITES

No Change in Weight or Girth Despite:
1. Sodium restriction to 20mEq/day
2. Water restriction to 1000ml/day
3. Intensive diuretic treatment:
 a. up to 400mg spironolactone qd
 b. up to 120mg furosemide qd
4. Urinary sodium remains < 10 mEq/day

tion is about 930 ml/24 hrs, a rate very similar to the maximum absorption rate of isotonic saline infused intraperitoneally into normal, noncirrhotic controls. Rapid diuresis can sometimes be tolerated if there is peripheral edema, but once this edema resolves, diuresis that induces a weight loss greater than one pound per day may do so by depleting the intravascular space and causing pre-renal azotemia. Thus, careful attention to daily weights is necessary. Refractory ascites *(Table III)* occurs in less than 5 per cent of patients with ascites. If the ascites is truly refractory, a peritoneovenous shunt may be considered.

Finally, once ascites is corrected, it is important to anticipate liberalized sodium intake at home and to adjust diuretics accordingly. The patient should be weighed daily, and any sudden weight gain of three to five pounds should prompt adjustment of the diuretic program until "dry weight" is achieved. Careful outpatient management can prevent recurrent hospitalization for ascites and its complications.

Self-Assessment Questions

1. Paracentesis is contraindicated in a patient with suspected bacterial peritonitis who has a severe coagulopathy. (T or F)
2. The most useful data from ascitic fluid analysis are specific gravity, protein, glucose, and cytology. (T or F)
3. Chylous ascites can be diagnosed by visual inspection. (T or F)
4. Hemorrhagic ascites implies tuberculous peritonitis. (T or F)
5. For ascites due to cirrhosis, the cornerstone of therapy is sodium restriction. (T or F)

(Answers on page 591)

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle if frequently used to keep the main title short.

Summary: all manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: the author is responsible for all statments made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

The Journal will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the Journal has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.



Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Drug Interactions With Oral Contraceptives

Question: Have there been reports of unwanted pregnancies caused by drug interactions involving oral contraceptives?

Answer: Oral contraceptives are among today's most commonly prescribed drugs. Oral contraceptive users, because of prolonged periods of ingestion, may receive concurrent drug therapy at some time during the period of contraceptive use. Drug interactions that diminish the efficacy of oral contraceptives should concern both clinician and patient. These interactions frequently involve rifampin, with at least 17 unwanted pregnancies reported,[1] and anticonvulsants which have led to at least 25 unwanted pregnancies.[2] Product information for some oral contraceptives[3] list drugs that may render them less effective. These lists include anticonvulsants, antibiotics, analgesics, tranquilizers, and antimigraine preparations. However, to date only two of these groups — anticonvulsants and antibiotics — have been reported to decrease the effectiveness of oral contraceptives leading to unwanted pregnancies. These drugs do so by one of two mechanisms.

Increased Metabolism

Drugs that may increase the metabolism of oral contraceptives include rifampin,[4-8] phenytoin, barbiturates, primodone, and carbamazepine.[2, 9, 10] Each of these agents is known to induce hepatic microsomal enzymes leading to increased metabolism of both estrogen and progesterones, thus decreasing the efficacy of the contraceptive and/or causing breakthrough bleeding.

Decreased Metabolism

Synthetic estrogens and progesterones are extensively excreted in bile, principally as glucuronide and sulfate conjugates.[11] Bacteria located in the gastrointestinal tract hydrolyze the conjugated estrogen leaving it free for reabsorption. Treatment with oral antibiotics would be expected to interfere with the hydrolytic process and thus reduce estrogen reabsorption from the gastrointestinal tract. Such a reduction in estrogen serum concentrations may decrease the efficacy of oral contraceptives. Unwanted pregnancies have been reported in women taking oral contraceptives in combination with ampicillin[12, 13] or tetracycline.[14] Chloramphenicol, nitrofurantoin, and sulfamethoxypyridine are also reported to decrease the efficacy of oral contraceptives, causing unwanted pregnancies and/or breakthrough bleeding.[15]

Summary

Unwanted pregnancies may result from drug interactions involving oral contraceptives and antibiotics (*e.g.*, ampicillin, tetracycline) that decrease the reabsorption of estrogens, or enzyme inducing agents (*e.g.*, rifampin, anticonvulsants) that increase the metabolism of contraceptive steroids. Simply increasing the estrogen dose may not be the answer to preventing pregnancies caused by these interactions. Special attention should be paid to spotting and breakthrough bleeding, which are signs of decreases in the estrogen serum concentration. Patients receiving oral contraceptives and short term antibiotic therapy should be advised to use additional forms of birth control. An alternative form of birth control may be appropriate for patients receiving long term anticonvulsant therapy.

Submitted by
Joyce Generali, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

PMIs

Drug Information for Patients

PHYSICIANS can now give their patients supplementary written instructions along with a prescription for any one of 20 of the most widely prescribed medications.

The American Medical Association has initiated a voluntary program that makes Patient Medication Instructions (PMIs) available to practicing physicians for appropriate distribution to patients. Use of the PMIs is expected to improve the effectiveness of therapy, reduce the risk of improper drug use, decrease the incidence of preventable and serious adverse drug reactions, and help patients to comply with instructions for taking their medications properly.

Now, more than ever, patients are seeking information about the drugs they are taking. The AMA welcomes patients' active interest in their own well-being and believes that a patient who understands why a drug has been prescribed is more likely to comply with his/her physician's directions. The AMA believes also that physicians are best qualified to provide reliable drug information that patients want and need. The PMIs offer physicians a method for presenting the information in a form that may be referred to by the patients when questions arise.

Available now are PMIs for 20 of the most widely prescribed drugs or drug classes. Physicians may obtain them from the AMA for a nominal charge that covers shipping and handling costs. PMIs for more of the commonly prescribed drugs and drug classes will be available next year. Eventually, the program will provide PMIs for as many as 100 drugs or drug classes and will represent the vast majority of all prescriptions written.

The format for each PMI is a 5½ x 8½ inch sheet printed on both sides. The instructions describe in clear, simple language the purpose of the drug, how it is to be taken, and its possible side effects — both minor side effects and those that should be reported to the physician. Space is provided to write in the dosage and any special instructions the physician may have for the individual patient. PMIs for individual drugs or drug classes are bound in pads of 100 sheets. They will be updated periodically when new information is available.

In a letter to some 400,000 practicing physicians (MDs and DOs), AMA Executive Vice President James H. Sammons, M.D., said of the PMI program, "It ranks among the most important programs the AMA has ever launched."

For the past decade, the medical community has generally agreed that patients need additional information about the drugs they take. Opinions have varied about the best method of supplying the information, however. The AMA has strongly supported the position that the most appropriate time to impart this information is during the physician-patient encounter.

The Food and Drug Administration (FDA) decided about a decade ago to require patient package inserts (PPIs) with oral contraceptives (the "pill"), and controversy has surrounded the PPI concept to some degree since then. The FDA in 1979 proposed rules that would have eventually required the dispensing by pharmacists of a PPI for virtually every prescription drug. Only 14 months later, however, this comprehensive plan was scaled down to a ten-drug pilot study. Then, in a 1981 hearing, FDA Commissioner Arthur Hull Hayes, Jr., M.D., received testimony that the private sector, especially the AMA, was taking steps to provide more information about prescription medications. In December 1981 it was announced that the mandatory PPI regulations would be rescinded.

Last May, Hayes wrote to Sammons to commend the inauguration of the AMA's PMI program. Terming the program "an important initiative in public information about drugs," Hayes said it is especially significant "because it provides for patients to receive written drug information directly from the prescribing physician." And, he wrote, "we at the FDA look forward to working with AMA in its efforts to get drug information to patients."

The PMI program is sponsored by the AMA Education and Research Foundation, which is seeking a broad base of financial support, including pharmaceutical firms and, later, possibly some foundations. Donations have already passed the $1.5 million mark. Groups that have provided financial support so far include Bristol-Myers Co.; Mead Johnson Foundation; Hoechst-Roussel Pharmaceuticals, Inc.; Hoffman-La Roche, Inc.; Johnson and Johnson; Eli Lilly and Co.; Revlon, Inc.; Upjohn Co.; Warner Lambert; American Home Products Corp.; Smith Kline and French Laboratories; and Burroughs Wellcome.

Although the AMA is investing in and is responsible for implementing the PMI program, it is drawing not only upon its own publication, *AMA Drug Evaluations,* but also on sources such as the *United States Pharmacopeia* for information. The United States Pharmacopeial Convention, Inc., is an independent, nonprofit corporation that sets official standards of purity, strength, and quality for drugs used in the United States. It additionally maintains a computerized information base on some 1,200 drugs.

PMI sheets are copyrighted by the AMA, but the association will permit physicians to use them "broadly for purposes of treatment." The sheets will indicate date of publication so that physicians can tell which of two sheets on the same drug is the more recent version.

Names and brief descriptions of the 20 drugs and drug classes in the first PMI series are listed below in no particular order of priority.

Furosemide: A very potent diuretic, effective in patients with impaired kidney function

Thiazide diuretics: Agents of choice as first line therapy in high blood pressure, useful for congestive heart failure in patients with normal kidney function

Penicillins (oral): Most wisely used antibiotics in treatment of many common bacterial infections

Digitalis preparations: Considered primary therapy for treatment of chronic congestive heart failure

Coumarin-type anticoagulants: Used in patients with blood clotting disorders and sometimes following strokes and heart attacks

Oral antidiabetics: Used for adult-onset diabetes and, when appropriate, can replace injectable insulin

Tetracyclines: Antibiotics used for many common bacterial infections and acne

Cephalosporins: Broad spectrum antibiotics useful for certain bacterial infections

Erythromycin: One of the safest antibiotics for treatment of bacterial infections

Nonsteroidal antiinflammatory agents: Useful for treatment of arthritis, to reduce inflammatory fevers, and pain

Benzodiazepines: Have almost replaced barbiturates for use as hypnotics; also widely used in anxiety

Sublingual nitroglycerin: Used under the tongue as drug of choice for treatment of chest pain

Methyldopa: Used in the treatment of high blood pressure

Insulin: Essential hormonal replacement for treatment of diabetes

Corticosteroids (oral): Have wide variety of uses, including reducing inflammation, treatment of arthritis, ulcerative colitis, and many more

Cimetidine: Inhibits secretion of stomach acid, and widely used in treatment of peptic ulcer

Belladonna & barbiturates: Widely employed for management of functional bowel syndromes

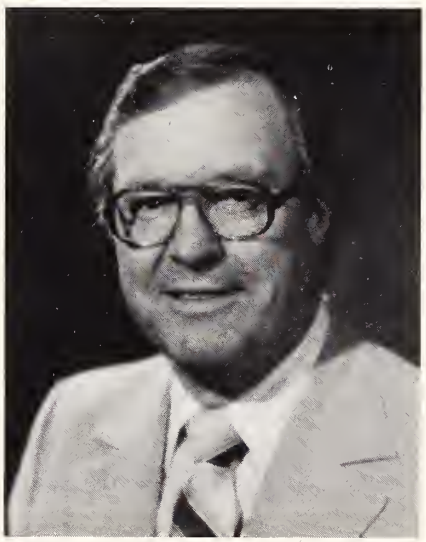
Phenytoin: A drug of first choice for treatment of many types of epilepsy

Sulfonamides: Widely used for treatment of urinary tract infections

Beta blockers: Used in the treatment of high blood pressure, chest pain, and heart beat irregularities

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

The President's Message



This Thanksgiving season I feel it is appropriate that we turn away from some of our problems of medicine and reflect on a Higher Being. This could be beneficial both to us and to our patients.

A Litany of Healing*

O God the Father, who desired all people to experience health and salvation;
HAVE MERCY UPON US.

O God the Holy Spirit, Who made our bodies to be temples of your presence;
HAVE MERCY UPON US.

We plead with you to hear us, O Lord; and grant Your grace to all who are sick, that they may be made whole;
WE BESEECH YOU TO HEAR US.

Grant to all who are disabled by injury or sickness, patience, courage, and sure faith in You, and give to all sick persons relief from pain, speedy healing, and fearless confidence in You;
WE BESEECH YOU TO HEAR US.

Grant to all about to undergo surgery Your strength, that they be not afraid;
WE BESEECH YOU TO HEAR US.

Give to all who are lonely or despondent, having no one to comfort them, the sense of Your presence;
WE BESEECH YOU TO HEAR US, O LORD.

Give to all doctors, nurses, and hospital personnel, Your wisdom, that with knowledge, skill, and patience they may minister to the sick with effectiveness;
WE BESEECH YOU TO HEAR US, GRACIOUS LORD.

Grant to the ministers of Your church and especially to chaplains in hospitals such grace, that what is done by their ministry may be perfected by Your power;
WE BESEECH YOU TO HEAR US, ALMIGHTY LORD.

You are the God that does wonders;
AND HAS DECLARED YOUR POWER AMONG THE PEOPLE.

For with You is the source of life;
AND IN YOUR LIGHT SHALL WE SEE LIGHT.

Turn us again, O Lord God of Hosts;
SHOW THE LIGHT OF YOUR COUNTENANCE, AND WE SHALL BE WHOLE.

We wait for Your loving-kindness, O God;
IN THE MIDST OF YOUR WORLD.

Sincerely,

Dennis J. Weidmer MD

President

* Used at the interfaith service of worship, Protestant Health & Welfare Assembly, St. Louis, March 11, 1980.



The Dubious Deification

Among the many admonitions directed at physicians regarding their conduct these days is the advice that they should avoid playing God. The direct approach can be a seeming accusation, not measurably relieved if it is converted to a question (Are you doctors still trying to play God?) since this approximates the old inquisitory impasse, have you stopped beating your wife? If one does attempt to pursue the matter with anything like objectivity, it becomes burdened with a variety of interpretations, utilizing terms and arousing thoughts of such variable content that there is no resolution agreeable to all. Hence, the specious profundity which makes it ideal for editorial comment.

First, there is the probability that if ours were not a primarily monotheistic culture, the reference would be to god rather than God which would change the concept considerably. The idea of a single god obviously endows that entity — and imitations thereof — with greater power than if it referred to one of the members of a board — Apollo, for example. Certainly, this puts a different connotation on the term but doesn't simplify the matter since an understanding of God's purposes has come to be many different things to different people and even so is subject to change with the circumstances. (As, for example, the engaging way in which the guardians of Islam have explained the adoption of some rather non-Islamic ways in the process of trying to get rid of $320 million a day by noting that it was God who put the oil under them in the first place.)

In addition to the numerous interpretations of God, there is also the consideration that the function of the physician, which is interpreted as usurpation of the god-purpose, is his essential discretionary role in altering the patient's existence for better or worse. But the physician is by no means alone in such effect. Legal-judicial-political agents, the bankers and other financial controllers, the clergy (although less than they would like) — virtually all of us in our most routine actions produce some influence on the lives of others as the social planners are eager to tell us. Why, then, are physicians singled out for special attention in this regard? Is it the not-uncommon armor of self-defense that is often taken for arrogance? Is it the association in the public mind of the physician with control over life and death, a control that has generally been less than the public likes to think? While there have been physicians who, with varying qualification, have capitalized on the patient's need for a god-dependent relationship, most have been content to perform as they saw fit, trying not to assume excess credit for their accomplishments but, at the same time, not disclaiming too vigorously any approval they may get for assisting God.

What this means is that such god-role as the physician has acquired (and is now being urged to reject) has not so much been sought as thrust upon him by a public longing for returned or retained health. The unsound spirit can be agonizing but those who work with it, if they are wise, know the risks of assuming identity with God and seek to avoid it. The spirit is, however, lacking in substance that can be the focus of a tangible form of expression or site of treatment. But the assaults upon the body by injury or disease, which the physician can generally alter rather visibly, prompt a more immediate identification of that physician with the sought-after (and God-given) salvation. After all, if the substance doesn't survive, spiritual survival will have to be transferred to whatever realm one's philosophy envisions, and that has rarely been seen by the subject as an attractive exchange except as a truly last resort. Even if the salvation is incomplete, the physician's efforts may suggest a god-like attempt at intervention — certainly not too far removed from God who has not always been consistent or understandable by human measure in his handling of things. And who can deny the therapeutic value of having a patient certain that his

medical attendant is God's own anointed? If the physician's efforts fail, the patient is not so apt to continue there as move on to another, hoping to find one more qualified for the deification that he will bestow when cured.

So once more, the irony of the physician's position is demonstrated. The physician, although accessory beneficiary to improved medical care, is also its victim. As medical successes have appeared, they have created not so much the relief of anxiety or comfort of secure health — after all, those things that are prevented from happening (and are thereby largely unknown) or are lost to the mind through short duration or prompt removal do not continue to press their urgency on the patient. They confirm the simple expectation that such things should not occur or, if they do, that they should not be allowed to cause the individual more than minimal inconvenience. Meanwhile, true medical successes have also resulted in the survival of many marginally productive individuals with a consequent distortion of the economy by creating an industry based on their maintenance. This is not a prelude to a recommendation for a "final solution" of eliminating such successes or their subjects, but a reminder that the reach will always exceed the grasp and it is in the process of reaching that the medical profession promotes the god-like image.

It is becoming more apparent that the complexity of medical service with increasing reliance on other agents and instruments dilutes the physician's direct involvement with the patient and leads to increasing attention to those intrinsic conditions that promote our physical and mental disabilities, to more concern about the extrinsic factors that have precipitated them and, in particular, to redirection of human activities so as to minimize the damaging effects.

This is, in fact, a much more human level of function — no reason to call upon God for such mundane purposes — and in passing we suggest this may account for some of the physician's distaste for some of the changes as well as the patient's disenchantment with a physician of only human stature (and the patient's willingness to turn to one or another of the faddists). At the least, a difficult task for the physician will be to convince the public that his role in such a scenario has much of any God-purpose. Prevention will be hard put to marshal anything like the public's fascination with miracle cures.

Despite well intended admonitions, then, we do not expect the physician to be released from the God role soon, whatever his personal efforts in that direction, as long as the patient values it. At the same time, the physician is reluctant to give up his place — not necessarily the God role but the dominance of the process in his own mind and, through it, the patient's mind. Since, in judicious doses, this image has been one of his most important therapies from earliest times, the idea of losing control or altering his sense of responsibility confronts him with an intolerable diminution. Even as he contends with the challenge, his efforts are often read as a continuing intent to play God. The patient's gratification with the attentions of the various agents and his extended survival still requires a focal point to which he can assign the God role. Perhaps this is why the continued complaints that their physicians, however knowledgeable and capable technically, fail to satisfy if they cannot reach the patient as an individual. The patient is not so much ready to release him in favor of these others as frustrated if he feels his physician contact is diminishing — and a similarity between the positions of the patient and physician becomes apparent. Whether the expectations of patients can be altered in the same sequence and rate as the adjustments of the physician's sense of purpose and method will determine whether the physician can satisfy his traditional role — whether God-like or human — in the new order.

The progress of the current efforts to condense medical service, to economize on costs by elimination of seemingly unnecessary or duplicative efforts, and to manipulate more "efficient" if less personalized use of physicians should tell us something about the state of the patient's attitude toward the profession — whether physicians are seen as God or gods or professional units — as the changes evolve. We suspect that much of the oft-cited annoyance of the public toward the traditional system of medical care is, in reality, an expression of this same frustration at the public level — how to retain the medical profession in its traditional role and still meet the costs. Physicians are fond of expressing the certainty that patients want their own individual physicians (God?), but perhaps the patient's attachment to a given physician has been partly from habit and partly from reluctance to change the scene. If the patient does accept the physician envisioned in the new order, it will promote changes more rapidly and there will be no need to warn physicians against playing God — the social methodology will take care of that.

There is, however, one specialized aspect of the admonition of professional concern. Physicians are urged to eschew the god-role lest their inability to satisfy its demands (or *their* interpretation of its

(Continued on page 591)

Vox Dox

Vox Dox Editor:

Third party reimbursement began in the 1930s when surgeons formed Blue Cross/Blue Shield to pay for their procedures because the cost was out of reach for the average patient. It is fair to say that currently the procedurally oriented specialties are still given preferential reimbursement for their services by Blue Cross/Blue Shield compared with reimbursement for cognitive services offered by other physicians including internists, pediatricians, psychiatrists, and family practitioners. A striking example of this is reflected in the fact that Kansas Blue Cross/Blue Shield with major medical benefits does not compensate for emergency medical treatment given on an outpatient basis, whereas full coverage including x-ray and laboratory studies is provided for acute surgical and traumatic emergencies.

There are many reasons for this. It is simpler from the insurer's standpoint to pay for the procedural services because they are more readily defined. It is difficult to establish an equitable reimbursement scale for cognitive services because they are subtler and less structured. The bulk of cognitive services, which includes preventive medicine, is rendered by the primary care specialties. While the public demand for cognitive services is increasing, the percentage of medical school graduates entering primary care has been drastically reduced since the late 1970s.[1] Theoretically all physicians should have a strong foundation in general clinical knowledge and skills, but certain subspecialties do not have such a base. If technological changes reduce the need for these subspecialties, physicians in these disciplines will be ill-equipped to return to primary care.[2] Meanwhile, income differential between physicians providing procedural services and those offering cognitive services continues to increase, creating even greater incentive for growth in the procedurally oriented specialties.

Procedural service specialists argue that non-procedural practitioners are envious because of their reduced monetary reimbursement. Further, procedural services physicians are of the opinion that they require more intensive education and carry a higher risk in delivering their care, and therefore are entitled to greater reimbursement.

President Reagan has proposed a $17.2 billion decrease in the Medicare budget of which 18 per cent will be cut from physician reimbursement.[3] Will this decrease in physician reimbursement affect primarily cognitive services? With the increased public demand for more preventive medicine, there will be a greater demand for cognitive services. If medicine becomes a technological profession, the need for physician services will decline and society will turn to other disciplines for cognitive services. Precedence for reimbursement for cognitive services is observed in other professions, such as law, banking, and engineering.

The outcome regarding the issue of equitable reimbursement for the different medical services will say much about our fundamental values. Until now, physicians have been considered experts in health care, but this notion becomes shaky as long as medicine as a profession devalues the delivery of and proper reimbursement for cognitive services. This issue, more than any other, has the potential to irreparably split the medical profession. Because this problem is national in scope, the American Society of Internal Medicine has written a White Paper on the subject that should be read by all physicians.[4] I accepted the Chairmanship of the Kansas Medical Society Ad Hoc Committee on Cognitive Medicine with the hope that the Committee will be able to generate fair solutions for more equitable reimbursement for cognitive services.

Joseph D. Sargent, M.D.
President
Kansas Society of Internal Medicine
P.O. Box 829
Topeka, KS 66601

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Vox Dox Editor:

The Kansas State Department of Education is requesting your assistance in appropriate recommendation for Homebound/Hospital programs in Kansas. Home and Hospital (H/H) instruction is an alternative delivery system which provides educational services outside of the school setting for physically or other health impaired children, and in some cases for children with personal and social adjustment problems.

Due to federal regulations (P.L. 94-142, 121a.550), the State of Kansas must assure to the federal government (1) that handicapped students will be educated with children who are not handicapped; and (2) that the removal of handicapped children from the regular educational environment occurs only when the nature or severity of the handicap is such that the use of supplementary aids and services cannot be achieved satisfactorily. In this manner, H/H programs are the most restrictive of all Special Education Programs. In addition, with H/H programs providing one teacher for each student, it is the most expensive of any program area.

For these reasons, extensive consideration should be given in recommending H/H for pregnancy. Your cooperation in bringing this matter to the attention of the physicians of Kansas is solicited to determine an appropriate period of time for which pregnant girls should be placed on H/H services. Since H/H programs are provided only to students who are *medically* unable to attend school, the State Department of Education has recommended that school districts limit H/H services to students who are pregnant to two months, unless extenuating circumstances (medically) exist.

Comments of Kansas physicians in regard to this matter would be most appreciated. The information obtained will be used in decisions to change present state rules and regulations. Please address your comments to me at the address below. If you should have questions, please contact my office (913) 296-3784. Thank you for your assistance.

Woody Houseman
State Dept. of Education
Home/Hospital Programs
120 East 10th
Topeka KS 66612

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The Dubious Deification

(Continued from page 589)

demands) leads to their own destruction. How does the physician retain a dominant and responsible position without taking on the stresses of omnipotence? This, we suggest, need not be interpreted as requiring total rejection of the role — a patent impossibility not free from possibly damaging results in itself — but an effort toward reorientation and determination of a balanced perspective. The problem here for physicians is that they like to make changes for themselves (or think they do) — not be pushed into them.

So, as we implied in the beginning, this exercise brings us no closer to resolution of the matter but one point remains valid. Let the physician play God — as long as he doesn't start believing it. — *D.E.G.*

Ascites

(Continued from page 583)

Answers

1. *False*
2. *False*
3. *False*
4. *False*
5. *True*

Selected Readings

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Renal Transplantation

(Continued from page 570)

The two most significant long-range problems in children with end-stage renal disease are growth failure and disruption of normal psychological and emotional development. Growth failure is a uniform finding in pre-pubertal patients with significant renal failure. Little catch-up growth is observed following renal transplantation, and generally the child follows the percentile at which he or she began at the time of transplantation. Growth potential seems to be somewhat better in patients transplanted before puberty. In addition, bone disease may be accentuated by steroids. Conversion to alternate-day steroid therapy may alleviate this problem to a limited extent.[9]

The psychosocial problems of children with severe renal disease may represent the most troublesome aspect of the illness.[10, 11] Because of their short stature, their dependence upon a machine for therapy, their requirement of continuous drug therapy, and because of their dependence upon physicians and hospitals, both the patients and their families find the situation a terrible ordeal. Therefore, the ideal approach to these patients requires a team including the physician trained in pediatrics and nephrology, social worker, psychologist, and nursing personnel. The possibility of loss of a transplanted kidney either through rejection or recurrence of the basic disease is always present and leads to an ever-present concern for the entire family. Constant counseling, advice, and support are mandatory if the patient and family are to adjust satisfactorily to the reality of this situation. We are continually amazed at how well children can adjust with proper guidance. A rather optimistic assessment of the final outcome of dialysis and transplantation in children can be obtained from the latest data of the European Transplant Registry for pediatric patients which shows that some 72 per cent of young patients who contracted chronic renal disease during childhood and who were treated with dialysis and/or transplantation, achieved complete rehabilitation with full active work capacity as adults eight years following the initiation of renal replacement therapy.[12]

At the present time, all of our patients are functioning very adequately and are able, with only minimal limitations, to take part in most normal daily activities. It is becoming clear that with modern therapeutic techniques, children with end-stage renal disease can often be rehabilitated to live a life with relatively few restrictions.

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The Ethical Context

(Continued from page 578)

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Aside from enabling patients to fall asleep more quickly and sleep longer, Dalmane seldom causes morning hangover. Most Dalmane patients feel alert and refreshed when they awaken. In 53 paired-night clinical studies comparing Dalmane and placebo in 2010 insomniac patients with a variety of secondary diagnoses, most Dalmane patients awakened more alert and refreshed, and less groggy and drowsy, than on nights when they had taken only placebo.[1] In a double-blind crossover study of 42 patients in private practice, approximately three times as many patients reported feeling refreshed and alert upon awakening after a night on Dalmane (flurazepam/Roche) compared to placebo nights.[2] This difference was highly significant ($p<0.001$). And a retrospective study of 2542 hospitalized patients who received Dalmane revealed only a 3.1% incidence of side effects.[3]

While residual effects from Dalmane therapy are infrequent, patients should be cautioned about drinking alcohol, driving or operating hazardous machinery after ingesting the drug.

Efficacy and safety in a broad range of patient types.

Over 2000 clinical trials involving more than 10,000 patients have shown that Dalmane patients fall asleep sooner, sleep longer and experience fewer nocturnal awakenings.[4] The safety and efficacy of Dalmane have been demonstrated in medical and surgical hospitalized patients, in patients seen in office practice and in elderly patients.[5-8] Since the risk of oversedation, dizziness, confu-

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sion and/or ataxia increases with larger doses in the elderly, it is recommended that the dosage be limited to 15 mg.

Moreover, the efficacy and safety of Dalmane for the treatment of insomnia have been demonstrated in thousands of patients with a variety of primary medical conditions, including cardiovascular, neuropsychiatric, endocrine-metabolic, gastrointestinal, genitourinary, respiratory and musculoskeletal disorders.[1] Dalmane (flurazepam HCl/Roche) is contraindicated in pregnancy and in patients hypersensitive to the drug.

Avoids rebound insomnia upon discontinuation.

Rebound insomnia—a worsening of sleep beyond pretherapy levels after drug discontinuation—has been reported as a potential clinical problem with some hypnotics.[9,10] However, this problem has not been reported with Dalmane. In eight out of eight sleep laboratory studies, there were no reports of rebound insomnia.[11] When you prescribe Dalmane, you can be confident of efficacy that enhances therapeutic progress. Your insomniac patients can be assured of a restful night, night after night—a good start for a good morning.

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Contraindications: Known hypersensitivity to flurazepam HCl, pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

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Obituaries

Frank A. Dlabal, M.D., 75, Wilson, The University of Kansas School of Medicine 1945, died July 11, 1982.

Frank A. Emery, M.D., 77, Wichita, Washington University School of Medicine 1929, died April 22, 1982.

Robert C. Goering, M.D., 56, Escuela Nacional de Medicina, Mexico, 1956, died April 16, 1982.

Pacharee Hompluem, M.D., 39, Parsons, Mahidol University, Thailand, 1969, died May 5, 1982.

Richard S. McKee, M.D., 76, Leavenworth, The University of Kansas School of Medicine 1935, died January 12, 1982.

Francis H. Relihan, M.D., 97, Smith Center, Northwestern University School of Medicine 1907, died March 10, 1982.

Clemens Rucker, M.D., 95, Topeka, Ensworth Medical College 1905, died May 21, 1982.

J. Colbert Simpson, M.D., 74, Salina, Creighton University School of Medicine 1938, died September 19, 1982.

Jack T. Stewart, M.D., 51, Wichita, Washington University School of Medicine 1957, died April 18, 1982.

Elmer G. Stolz, M.D., 64, Wichita, Creighton University School of Medicine 1945, died October 25, 1981.

Vernon C. Wiksten, M.D., 70, Topeka, The University of Kansas School of Medicine 1937, died January 19, 1982.

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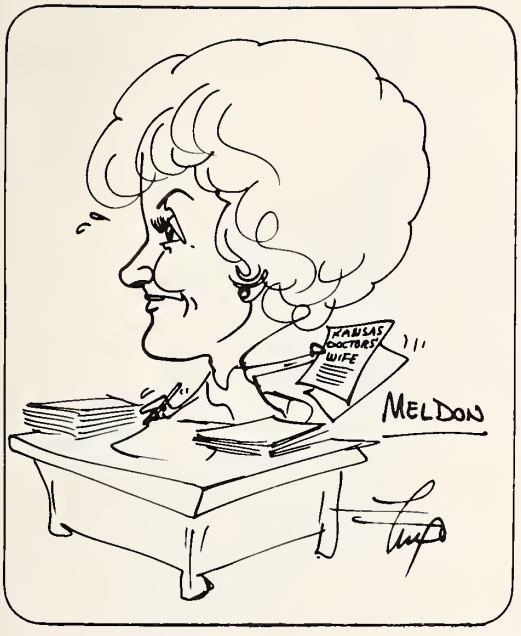
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AUXILIARY NEWS

An Open Letter to Kansas Physicians

Dear Doctor:

The annual "Confluence" of the AMA Auxiliary, a three-day leadership training conference, was held in Chicago last month. Representing Kansas were KMSA President Meldon Laury; President-Elect Diane Sanders; Membership Chairman Caryl Bichlmeier; and four county presidents-elect: Linda Ellison, Salina; Mary King, Great Bend; Mary Belle Boyd, Wichita; and Ora Alexander, Kansas City.

Presentations on time management, cooperation with medical societies, intergenerational problems of the aging, and the mid-life problems in the medical family were all interesting and well received. But the most dynamic and dramatic part of the seminar was on drug abuse which included four speakers.

The Director of the Parent Resources Institute for Drug Education (PRIDE), Thomas Gleaton, Ed.D., asks that physicians submit case histories on the effects of drugs. He suggests that the physician can be instrumental in effecting cures in the drug abuse epidemic among youth much as they have fought polio. Please send your case histories to Dr. Gleaton, 930 Crestline Road NE, Alanta GA 30328.

Marsha Schuchard, Ph.D., Atlanta, author of *Parents, Peers and Pot,* made the greatest impact on the audience with her presentation. She stated that the illegal drug trade in this country is the biggest money maker in the world. People in other countries are astounded that parents in the United States allow their children to live in a world that makes drugs an acceptable part of life, with peer pressure so strong that to say "no" to drugs is considered rude. Dr. Schuchard stated that television and movies are a particularly bad influence on youth, presenting the use of illicit drugs as acceptable and fun. Included in this workshop was an array of drug paraphernalia, including a frisbie with an inhalation gadget (the first to get stoned wins); a T-shirt ("A Day Without Dope Is a Day Without Sunshine"); and other items demonstrating that the deviant is now the norn.

After attending this drug abuse seminar, the Auxilians are more than ever dedicated to our campaign directed against drug abuse. We have "Fonz and Friends" puppets with a script about drug abuse available for our members to present to grade school children, along with a free "Dirty Dozen" brochure which will be distributed across Kansas. Our "STOP POT" bumper stickers are selling well, and can be ordered from the Kansas Medical Society, three for $1. We hope you will put one on your car.

The state-wide drug abuse conference scheduled for April 8-9, 1983, in Salina, co-sponsored by the Auxiliary and KMS, will be a major event. Other participants in the conference will be the Social and Rehabilitation Services (SRS); Kansas State Nurses Association; Kansas Pharmacists Association, State Department of Education; Kansas High School Activities Association, Department of Health/Environment; and others. Please mark your calendar now for this important event.

Meldon Laury
President
Kansas Medical Society Auxiliary

RESOURCE FOR PHYSICIANS IN TROUBLE

The Kansas Medical Society Impaired Physicians Program is now operational. If you desire more information concerning this program, if you know an impaired colleague who needs help, or if you are concerned about yourself or your spouse, please contact one of the Committee members nearest you, as listed below, or the KMS Executive Office. All such contacts will be held in strictest confidence and the caller need not reveal his name, if he/she so desires.

Alcoholism, other drug abuse, and medical/neurological/psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

John Cody, M.D., Hays (913) 628-2871
Robert P. Hudson, M.D., Kansas City ... (913) 588-7040
H. Ivor Jones, M.D., Shawnee Mission .. (913) 362-4040
Mary L. Rardin, M.D., Wichita (316) 688-3070
Ivan Rhodes, M.D., Wichita (316) 685-1291
Alex Scott, M.D., Junction City (913) 238-2518
Max Teare, M.D., Garden City (316) 276-7689
David L. Trudeau, M.D., Wichita (316) 685-1111
Donald R. Tucker, M.D., Topeka (913) 354-5275
Virginia L. Tucker, M.D., Lawrence (913) 862-9360 Ext. 215
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CONTRAINDICATIONS: Patients hypersensitive to ibuprofen, or with the syndrome of nasal polyps, angio-edema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory drugs (see WARNINGS).

WARNINGS: Anaphylactoid reactions have occurred in patients hypersensitive to aspirin (see CONTRAINDICATIONS). Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Peptic ulceration, perforation, or gastrointestinal bleeding can end fatally, however, an association has not been established. Rufen should be given under close supervision to patients with a history of upper gastrointestinal tract disease, and only after consulting the ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be attempted. If Rufen must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

PRECAUTIONS: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If developed, discontinue Rufen and administer an ophthalmologic examination.

Fluid retention and edema have been associated with Rufen; caution should be used in patients with a history of cardiac decompensation.

Rufen can inhibit platelet aggregation and prolong bleeding time. Use with caution in patients with intrinsic coagulation defects and those taking anticoagulants.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy, this therapy should be tapered slowly when adding Rufen.

DRUG INTERACTION: *Coumarin-type anticoagulants.* The physician should be cautious when administering Rufen to patients on anticoagulants.

Aspirin. Concomitant use may decrease Rufen blood levels.

PREGNANCY AND NURSING MOTHERS: Rufen should not be taken during pregnancy nor by nursing mothers.

ADVERSE REACTIONS: Incidence greater than 1%. Gastrointestinal: The most frequent adverse reaction is gastrointestinal (4 to 16%). Includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). Central Nervous System: dizziness*, headache, nervousness. Dermatologic: rash* (including maculopapular type), pruritus. Special Senses: tinnitus. Metabolic: decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS). *Incidence 3% to 9%.

Incidence less than 1 in 100. Gastrointestinal: gastric or duodenal ulcer with bleeding and/or perforation, hemorrhage, melena. Central Nervous System: depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. Dermatologic: vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome and alopecia. *Special Senses:* hearing loss, amblyopia (blurred and/or diminished vision, scotomata and/or changes in color vision) [see PRECAUTIONS]. Hematologic: neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs' positive), thrombocytopenia with or without purpura eosinophilia, decreases in hemoglobin and hematocrit. Cardiovascular: congestive heart failure in patients with marginal cardiac function, elevated blood pressure. Allergic: syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasms (see CONTRAINDICATIONS). Renal: acute renal failure in patients with preexisting significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. Miscellaneous: dry eyes and mouth, gingival ulcers, rhinitis.

Causal relationship unknown. Gastrointestinal: pancreatitis. Central Nervous System: paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. Dermatologic: toxic epidermal necrolysis, photoallergic skin reactions. Special Senses: conjunctivitis, diplopia, optic neuritis. Hematologic: bleeding episodes. Allergic: serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. Endocrine: gynecomastia, hypoglycemia. Cardiovascular: arrhythmias (sinus tachycardia, bradycardia, and palpitations). Renal: renal papillary necrosis.

OVERDOSAGE: Acute overdosage, the stomach should be emptied. Rufen is acidic and excreted in the urine, alkaline diuresis may benefit.

DOSAGE AND ADMINISTRATION: Rheumatoid arthritis and osteoarthritis, including flareups of chronic disease: Suggested dosage 400 mg t.i.d. or q.i.d.

Dysmenorrhea: 400 mg every 4 hours as necessary.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for the relief of pain. *Do not exceed 2,400 mg per day.*

CAUTION: Federal law prohibits dispensing without prescription.

Council Meeting

Report of Meeting Held September 26, 1982

A meeting of the Council was held on Sunday, September 26, 1982, at the Holidome-Topeka, beginning at 10 AM. Kermit G. Wedel, M.D., President, presided. Present were: Drs. Franklin G. Bichlmeier, F. Calvin Bigler, James G. Bridgens, K. William Bruner, Jr., Thomas P. Butcher, Clair C. Conard, Louis M. Culp, Robert D. Durst, Jr., Herbert Fransen, Jimmie A. Gleason, Robert J. Haskins, Robert W. Hughes, John Neuenschwander, Sr., Lucien R. Pyle, James H. Ransom, Edwin D. Rathbun, Alex Scott, Newton C. Smith, Marvin Snowbarger, Wayne Wallace, Jr., Wallace N. Weber, and Emerson D. Yoder. Also present were: J. G. Hollowell, M.D., Mrs. Meldon Laury, Mrs. Kathleen Wedel, Steve Carter, Gary Caruthers, and Val Braun.

The Council took the following actions:

Mediserve

- That Mediserve, Inc., not consider new applicants;
- That KMS loan to Mediserve $10,000 in 1983 and $10,000 in 1984;
- That KMS discontinue all loans and contributions to Mediserve after 1984;
- That Mediserve begin repayment of KMS loans as soon as students begin to repay loan principal and interest.

Consolidation of Funds

The remaining separate KMS funds — Journal and State Meetings — will be combined with the General Fund. In the future, funds will be itemized as budgeted line items.

Kansas Council on Economic Education Grant

Funding for this project of tuition to teachers enrolling in economic education classes is considered a one-time grant, not to be refunded in 1983.

Salaries

The Council granted the staff a cost-of-living/merit salary increase of 10 per cent, and recommended that David E. Gray, M.D., be compensated for his present duties as *JKMS* Editor.

Reserves

Establishment of reserves at a minimum of 25 per cent of the operating budget was approved. Once the reserve level is reached, it would not be used for routine operating expenditures without the approval of the Executive Committee and Council.

Impaired Physician

The Council reiterated its support for the impaired physician project. The KMS will loan to that program $15,000 on January 1, 1983, as seed money for the Kansas Impaired Physician Fund, a separate corporation which will be activated on that date. The Council deferred all other requests for funds for this program, including the Impaired Physician Fellowship, indicating that they should be considered for funding from the Kansas Impaired Physician Fund.

Dues

The 1983 dues were set at $180. A comparison of dues levels showed that the median dues level in 1983 would be $242. The 1983 dues for comparable states were: Colorado — $330, Iowa — $275, Arkansas — $225, Nebraska — $245, Oklahoma — $210, and Missouri — $125 (Missouri receives a substantial portion of its revenue from a self-insurance fund). KMS dues were last increased in 1978 (for 1979). Nationally, Kansas dues ranked 47th. The current dues increase approval carries with it a recommendation that dues be reviewed on an annual basis, in the belief that incremental adjustments, if needed, are preferable to periodic larger increases.

1983 Annual Session

The meeting, formerly scheduled for Topeka, has been moved to Holiday Inn-Holidome, Lawrence. The meeting will be hosted by the Douglas County Medical Society. The reason for the move was that the Ramada Inn-Topeka would not accommodate KMS because of another larger meeting. Staff was directed to express dismay at the manner in which the KMS meeting arrangements were handled by the Ramada Inn.

FTC Legislation

Drs. Wedel and Gleason and Steve Carter recently visited with the Kansas Congressional representatives in Washington, D.C. on the issue of continued

funding for the FTC and the FTC control over the professions. The two bills included the Luken-Lee bill that would place a moratorium on FTC action regarding state-regulated professions until Congress expressly directs it to act. An alternative amendment offered by Rep. James Broyhill (D-N.C.) would mandate FTC jurisdiction over the professions and would permit the agency to preempt state laws concerning professionals.

Legislative Update

Jerry Slaughter, Governmental Liaison, reported a resolution recently adopted by the Kansas Board of Healing Arts that allows chiropractors to use the term "physician" in their advertising. KMS Legal Counsel rendered an opinion indicating that there is no statutory provision to allow the use of such term. KMS Executive Committee approved requesting that the Board of Healing Arts reconsider their action on this issue.

In considering the issue of Certificate of Need, one provision would require the purchase of any major medical equipment by anyone to be approved by the CON process. KMS is on record as opposing this issue. The federal government is currently considering action either to discontinue or alter CON and the Health Planning Laws.

An interim legislative committee is studying the issue of Advanced Registered Nurse Practitioners, and whether to revise the statute in view of the recent court finding in favor of the KMS position. KMS presented a proposal to ensure that the physician would remain the director of the health care team.

Another issue being considered in the legislature is that of self-insuring health insurance for state employees. The Committee is considering the possibility of bidding state business out on a fee-for-service basis for the fee schedule or on a prepaid HMO basis.

KMS Auxiliary

Mrs. Meldon Laury (David) thanked KMS for the $4,000 in financial support provided to the Auxiliary by the KMS. She reported that Auxiliary dues will remain at $7 for 1983. The Auxiliary is active in the area of drug abuse. The STOP POT! bumper stickers are available through local Auxiliary chapters; a puppet show on the use and abuse of marijuana has been developed for presentation in public schools; and the Auxiliary is participating in the planning of the statewide drug abuse forum scheduled for April 8-9, 1983.

The meeting adjourned at 12:30 PM.

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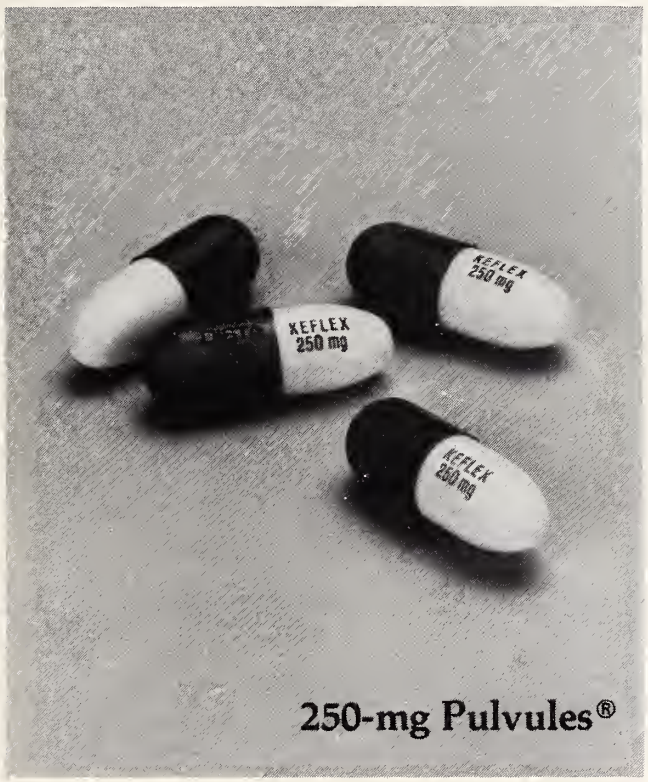
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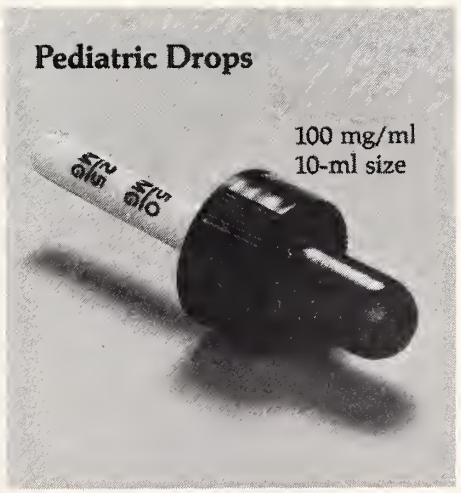
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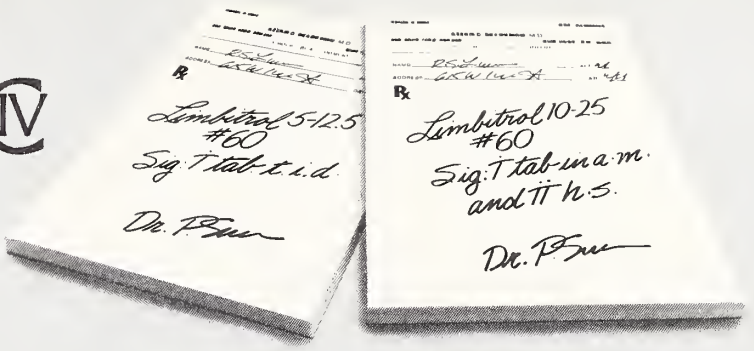
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References: 1. Clagham J: *Psychosomatics 11*:438-441, Sept-Oct 1970. **2.** Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine*, edited by Jarvik ME. New York, Appleton-Century-Crofts, 1977, p. 316. **3.** Baldessarini RJ, Tarsy D: Tardive dyskinesia, in *Psychopharmacology: A Generation of Progress*, edited by Lipton MA, DiMascio A, Killam KF. New York, Raven Press, 1978, p. 999.

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
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Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

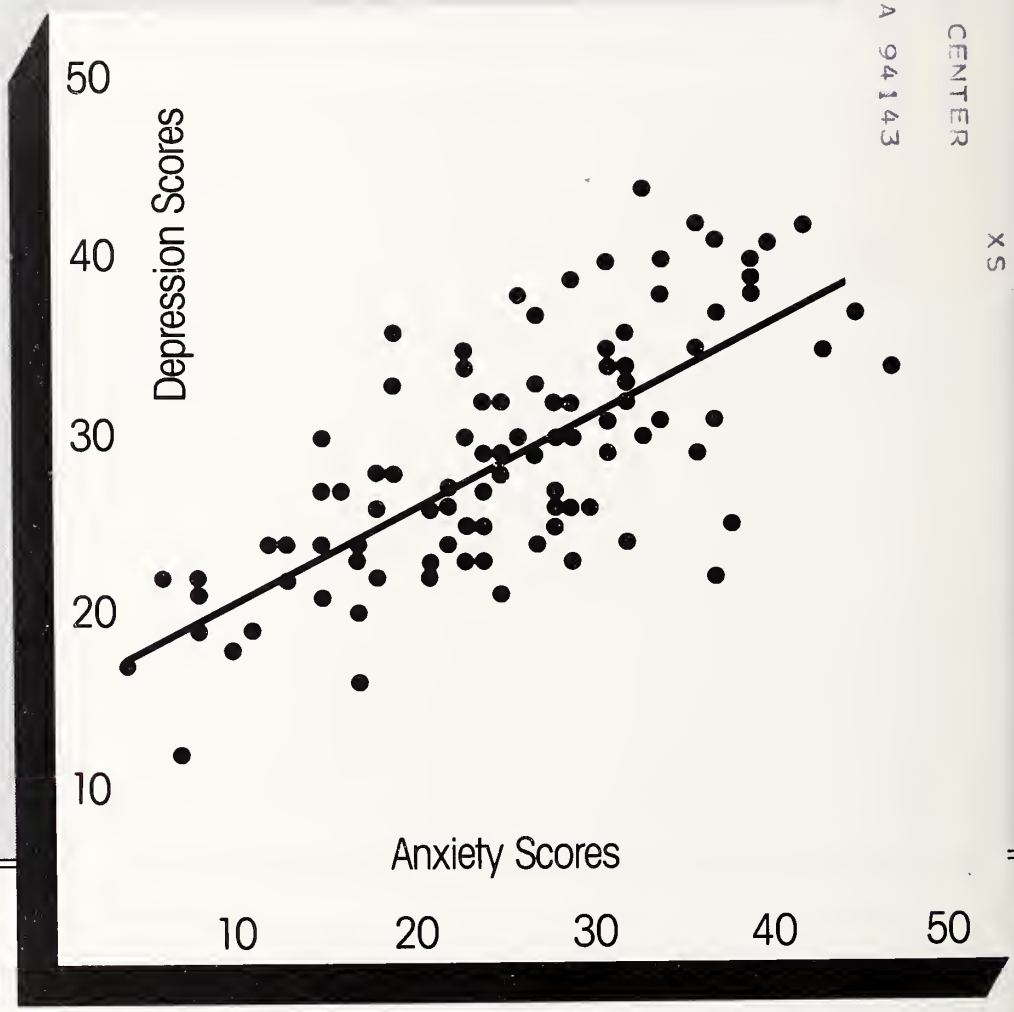
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
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Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
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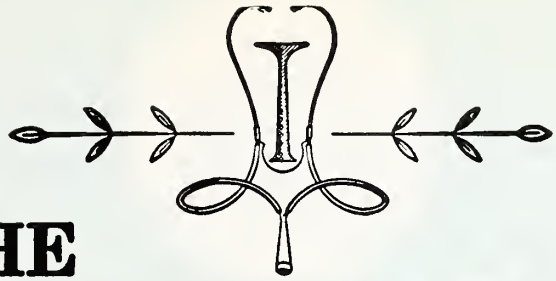
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Kermit G. Wedel,
President
Gary Caruthers
Val Braun
The coming of Christmas signals the end of another busy year. We hope 1982 has been pleasant and generous to you and your family. This is a good time for each of us to reflect on our good fortunes and to renew our dedication and responsibility to our profession, family, and patients. The staff and officers of the Kansas Medical Society wish each of you a joyous holiday season!
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ANUSOL-HC® Suppositories/ ANUSOL-HC® Cream

Before prescribing, please see full prescribing information. A Brief Summary follows:

Indications and Usage: Anusol-HC Suppositories and Anusol-HC Cream are adjunctive therapy for the symptomatic relief of pain, itching and discomfort in external and internal hemorrhoids, proctitis, papillitis, cryptitis, and fissures, incomplete fistulas, pruritus ani and relief of local pain and discomfort following anorectal surgery.

Anusol-HC is especially indicated when inflammation is present. After acute symptoms subside, most patients can be maintained on regular Anusol® Suppositories or Ointment.

CONTRAINDICATIONS

Anusol-HC Suppositories and Anusol-HC Cream are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparations.

WARNINGS

The safe use of topical steroids during pregnancy has not been fully established. Therefore, during pregnancy, they should not be used unnecessarily on extensive areas, in large amounts or for prolonged periods of time.

PRECAUTIONS

General

Symptomatic relief should not delay definitive diagnoses or treatment.

Prolonged or excessive use of corticosteroids might produce systemic effects.

If irritation develops, Anusol-HC Suppositories and Anusol-HC Cream should be discontinued and appropriate therapy instituted.

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled.

Anusol-HC is not for ophthalmic use.

Pregnancy

See "WARNINGS"

Pediatric Use

Care should be taken when using the corticosteroid hydrocortisone acetate in children and infants.

DOSAGE AND ADMINISTRATION

Anusol-HC Suppositories—Adults: Remove foil wrapper and insert suppository into the anus. Insert one suppository in the morning and one at bedtime for 3 to 6 days or until inflammation subsides. Then maintain comfort with regular Anusol Suppositories.

Anusol-HC Cream—Adults: After gentle bathing and drying of the anal area, remove tube cap and apply to the exterior surface and gently rub in. For internal use, attach the plastic applicator and insert into the anus by applying gentle continuous pressure. Then squeeze the tube to deliver medication. Cream should be applied 3 or 4 times a day for 3 to 6 days until inflammation subsides. Then maintain comfort with regular Anusol Ointment.

NOTE: If staining from either of the above products occurs, the stain may be removed from fabric by hand or machine washing with household detergent.

Store between 59°-86°F (15°-30°C)

1089G010

Tax Equity Act

Proposed Federal Rules on Reimbursement for Physician Services

On November 13, 1982, Kermit Wedel, M.D., President of the Kansas Medical Society, submitted comments in opposition to the new proposed rule concerning the establishment of criteria and maximum limits on reimbursement for physician services furnished to Medicare beneficiaries by "provider-based" physicians.

In objecting to this rule, KMS joined the AMA and other medical organizations in indicating the rule was inappropriate and would adversely affect medical practice. KMS further urged the Health Care Financing Administration to withdraw and rewrite the proposal.

The proposed regulations would differentiate between those physician services that are furnished to individual patients and those that are provided to the general patient population of an institutional provider. To accomplish this, hospitals and other facilities that compensate physicians would have to allocate the percentage of "physician services to the provider" and "physician services to patients." All physicians' services would be presumed to be Part B (for benefit of individual patients) unless a written time allocation agreement is in force between the hospital and the physician. The proposal would require detailed record-keeping to verify the allocations. Physician compensation costs would be defined to include:

> Monetary payments, fringe benefits, deferred compensation and any other items of value (excluding office space related to treatment of private patients, or billing and collection services) a provider or other entity (such as a medical school or other organization related to the provider) furnishes a physician in return for the physician's services.

The Health Care Financing Administration (HCFA), through application of the reasonable compensation equivalents (RCE), intends to establish a limit for the amount of total Medicare compensation that a provider-based physician may receive for *both* physician services to providers and physician services to individual patients. More specifically, the regulations would authorize Part B reimbursement only in situations where:

- The services are personally furnished for an individual patient by a physician;
- The services ordinarily require performance by a physician, and are not frequently and consistently furnished by non-physicians; and
- The services must contribute directly to the diagnosis or treatment of an individual patient. (Additional requirements must also be met for Part B reimbursement for anesthesiology, radiology, and laboratory services.)

Services that provider-based physicians render that do not meet the above requirements and are related to the provision of patient care by the institution are to be reimbursed under Part A reasonable cost rules. In situations where a physician enters into an agreement whereby he or she assumes all of the operating costs of provider's department, the physician would be entitled only to reasonable charge payment for services to individual patients, but the institution would be entitled to recovery for incurred costs associated with the physician services.

Anesthesia Services

Under the proposed rule, Part B reimbursement to anesthesiologists would be authorized for up to two concurrent anesthesiology services. Where the physician is supervising more than two concurrent procedures or does not meet all of the criteria listed below, those services in concurrent cases will be considered to be for the benefit of the institution and will be reimbursed under Part A. To be eligible for Part B charge reimbursement in concurrent cases, for each patient the physician must:

- perform a pre-anesthetic examination and evaluation;
- prescribe the anesthesia plan;
- personally participate in the most demanding procedures in the anesthesia plan, including induction and emergence;
- ensure that any procedures in the anesthesia plan that he or she does not perform are performed by a qualified individual;
- monitor the course of anesthesia administration at frequent intervals;
- remain physically present and available for immediate diagnosis and treatment of emergencies; and

(Continued on page 630)








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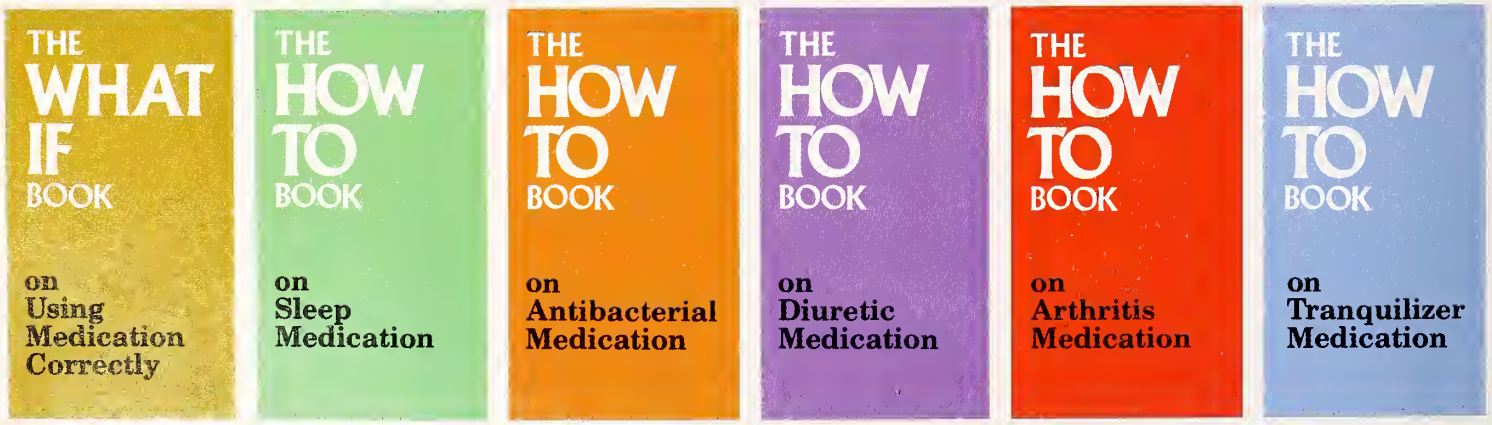
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The Kansas Medical Political Action Committee was formed by the Kansas Medical Society in 1962 in order (1) to promote and strive for the improvement of government by encouraging and stimulating physicians and others to take a more active and effective part in governmental affairs; (2) to encourage physicians and others to understand and evaluate the nature and actions of government, the significance of political issues, the records of officeholders, and the pertinent qualifications of candidates for elective office; and (3) to assist physicians and others to organize themselves for effective political action to promote civic responsibility and integrity in government.

The Kansas Medical Society has a membership of 2,783 and the Kansas Medical Auxiliary has a membership of 1,330. The following physicians and spouses have elected to become members of KaMPAC and to support "organized medicine" in its efforts to elect "friends of medicine." This listing will be expanded each month to include new KaMPAC members.

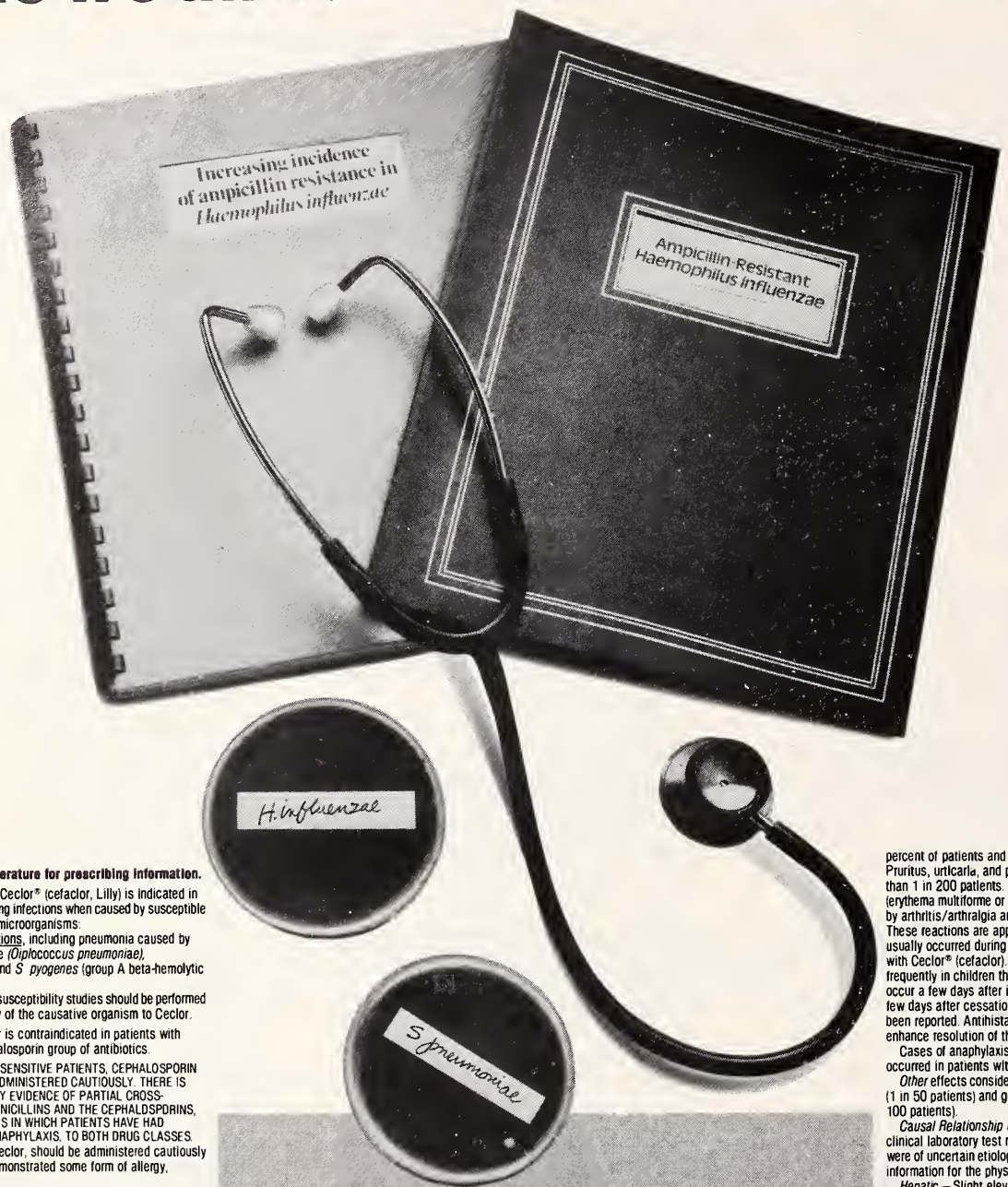
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An added complication... in the treatment of bacterial bronchitis*



Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor
Pulvules®, 250 and 500 mg

Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae,* and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coomb testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: Adverse effects considered related to cefaclor therapy are uncommon and are listed below:

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70) and nausea and vomiting (1 in 90).

As with other broad-spectrum antibiotics, colitis, including rare instances of pseudomembranous colitis, has been reported in conjunction with therapy with Ceclor.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor® (cefaclor). Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [100281R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.[8]

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

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8. Principles and Practice of Infectious Diseases (edited by G.L. Mandell, R.G. Douglas, Jr., and J.E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285.

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(Continued on page 610)

Medicament Predicament.



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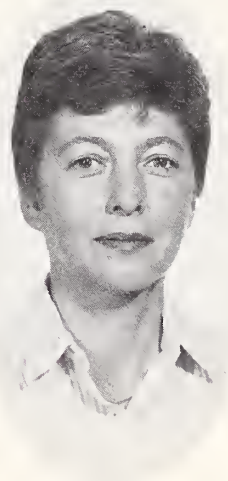
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(Continued from page 607)

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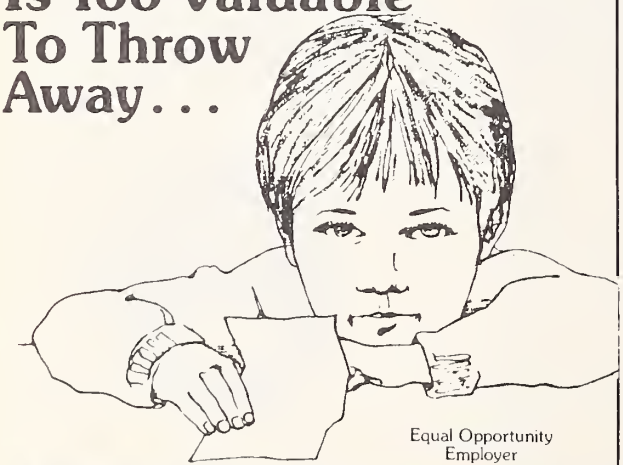
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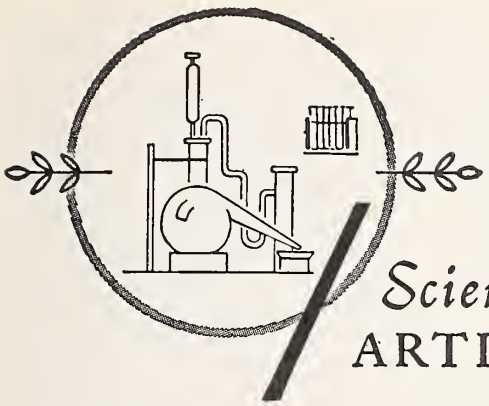
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Percutaneous Transluminal Angioplasty

The Treatment of Stenotic or Occluded Superficial Femoral Arteries

JOSEPH P. GALICHIA, M.D.; GREGORY F. DUICK, M.D.;
ASHOK K. BAJAJ, M.D. *and* **ROGER W. ROBERTS, D.O.** *Wichita*

Percutaneous transluminal angioplasty was performed in 30 patients with 46 superficial femoral artery lesions. Initial successful dilatation was obtained in 33 (89.2%) of the 37 superficial femoral stenoses, and six (66.7%) of the nine occluded superficial femoral arteries. This procedure is an effective alternative to surgery, especially important for medically complex patients and those refractory to surgery itself.

PERCUTANEOUS transluminal angioplasty (PTA) utilization has increased exponentially in this country during the last four years. PTA has been employed in relieving obstructions in the coronary, iliac, femoral, renal, superior mesenteric, and subclavian arteries.[1, 2] The potential use of PTA is being expanded to include renal angio-access shunts and previous surgical bypass grafts.[3] Recently, PTA has also been used to treat carotid artery stenosis and even basilar artery stenosis with good success.[4, 5] PTA can be used on a variety of lesions causing vascular ischemia, including atherosclerosis, thrombosis, fibromuscular dysplasia, and Takayasu arteritis.[6]

From February 1980 to March 1982 we have used the Grüntzig method for dilatation of 46 highly stenosed or totally occluded superficial femoral arteries in a total of 30 patients. PTA was attempted as treatment in these patients with disabling claudication, rest pain, or early signs of gangrene. All patients had indications for femoro-popliteal bypass grafting, but many were poor surgical candidates. Non-invasive studies, aorto-femoral angiograms, and surgical consults were obtained prior to PTA. Our patients included 20 men and ten women, ranging in age from 47 to 84 years, with a mean of 66.9 years *(Table I)*. Initial successful dilatation was defined as improvement of the arterial narrowing or occlusion by angiography, post-PTA, and improvement of clinical symptoms during the hospitalized course.

Methodology

Satisfactory angiograms of the aorta, common iliac, and femoral vessels were obtained for each patient. Dilatation of the superficial femoral artery is performed by an antegrade puncture of the common femoral artery, approximately two cm above the inguinal ligament. A J-tipped guide wire and an angiographic catheter over the wire are introduced proximal to the lesion. The guide wire is advanced across the stenosis or total occlusion in a J-tipped configuration, when possible. The angiographic catheter is then also passed across the lesion over the

From the Department of Cardiology, St. Francis Hospital, UKSM-Wichita.

TABLE I
PATIENT RISK FACTOR DISTRIBUTION

	Number	*Mean Age*	*Smokers*	*Hypertension*	*Diabetes Mellitus*
Male	20	65.5	15 (75.0%)	10 (50.0%)	5 (25.0%)
Female	10	69.7	4 (40.0%)	9 (90.0%)	5 (50.0%)
Total	30	66.9	19 (63.3%)	19 (63.3%)	10 (33.3%)

guide wire, and distal pressure is measured. The angiographic catheter is exchanged for a Grüntzig dilating catheter, which is repeatedly inflated to four atmospheres of pressure. In the case of totally occluded superficial femoral arteries, the dilatation is performed segmentally, proceeding from distal to proximal. Intraluminal pressures upon pullback and a post PTA angiogram are obtained to evaluate the degree of success. After dilatation has been completed, hemostasis is assured at the puncture site. The patient is kept at bedrest for six hours and intravenous heparin administered for 12-48 hours. Aspirin and dipyridamole (Persantine) are administered following the procedure and continued for six months after PTA.

Case Report

A 64-year-old white female presented with a history of mild burning sensation in both feet for three years. She stated that she had developed a new pain in the right lower thigh and calf during the last eight to nine months, which markedly worsened upon 70-80 feet of walking. She had a 180 pack-year history of smoking, diabetes for five years, hypertension, and obesity. She also had a medical history of atherosclerotic heart disease, congestive heart failure, five past myocardial infarctions, and chronic bronchitis. Upon physical examination, she was found to have weak popliteal pulses bilaterally. Posterior tibial and dorsalis pedis pulses were absent on the right side. A non-invasive study revealed a hemodynamically significant lesion of the right superficial femoral artery. A nerve conduction study revealed decreased nerve conduction velocity of the right posterior tibial nerve. It was felt that the patient had bilateral diabetic neuropathy and an overlapping pain of ischemic origin on the right side. An arteriogram showed a 5 cm total occlusion of the middle superficial femoral artery *(Figure 1)*. The patient was treated with PTA, and a post-PTA angiogram showed patency of the native superficial femoral vessel *(Figure 2)*. She continued to have the mild burning pain in her feet, but the claudication pain in the right calf and the lower thigh improved dramatically.

Results

Immediate results of PTA treatment of the 46 lesions in 30 patients are summarized in *Table II*. Of the 37 superficial femoral artery stenoses, we were



Figure 1. Before PTA there is a five cm total occlusion of right superficial femoral artery.

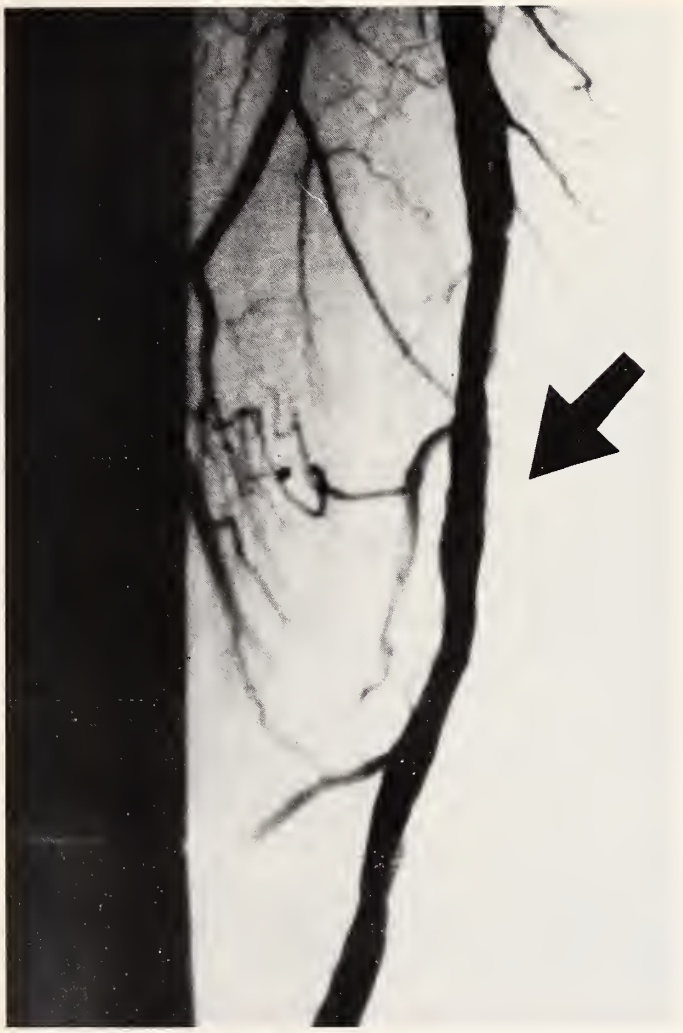


Figure 2. After PTA, the artery is patent at the occlusion site and the caliber of the vessel proximally and distally is markedly improved.

able to successfully dilate 33 (89.2%) vessels and six (66.7%) of the nine totally occluded superficial femoral vessels. We were unable to cross three vessels — one severe stenosis and two total occlusions — due to vessel tortuosity, extreme narrowing, or occlusions from hardened plaques. In one patient, there was poor distal run-off post satisfactory dilatation of the lesion itself. One patient had a 20 cm occlusion of a femoral-popliteal artery, and PTA was used as a salvage attempt to prevent amputation. This was unsuccessful due to inability to cross the entire occlusion with poor distal run-off.

During angioplasty, several radiopaque fragments were seen to embolize distally in one patient with superficial femoral stenosis. The catheter was left in place and the patient was treated with overnight streptokinase infusion. The next morning, the great toe was cyanotic, but this resolved during the hospitalization with improvement of the patient's claudication symptoms. Another patient with superficial femoral artery stenosis underwent a successful dilatation, but the distal flow was obstructed at the trifurcation, secondary to an acute thrombosis. The femoral catheter was left in place and he was treated with overnight streptokinase infusion, with successful resolution of the thrombus and good distal flow.

Discussion

The results of dilatation of the superficial femoral vessels in our series compares similarly to the work of others. We were most successful in treating short stenotic lesions in the superficial femoral arteries. The work of others shows an initial success rate between 72 and 100 per cent in superficial femoral artery stenosis, and 28 to 100 per cent for superficial femoral occlusions.[7-11] Others have experienced problems with long occluded segments, such as in our patient with a 20 cm long occlusion in the femo-

TABLE II
SUPERFICIAL FEMORAL ARTERY LESIONS: RESULTS AND COMPLICATIONS

	Catheterization Attempted	*Inability to Cross Lesion*	*Balloon Dilatation Attempted*	*Initial Successful Dilatation (of All Patients)*	*Poor Distal Run-Off*	*Distal Embolization*	*Acute Distal Thrombosis*
Superficial femoral stenosis	37	1	36	33 (89.2%)	1	1	1
Superficial femoral occlusion	9	2	7	6 (66.7%)	1	0	0
Total	46	3	43	39 (84.8%)	2	1	1

ral popliteal system.[10] Previously reported complications of PTA include distal embolization, acute thrombosis, vessel perforation, balloon rupture, and hematoma at the site of arterial puncture.

Spence *et al.* have followed 103 femoral artery lesions for two years.[9] Of the initially successful dilated lesions, they noted a patency rate of 79.6 per cent after one year, and 74.9 per cent after two years. Comparing this with femoro-popliteal bypass surgery, Naji *et al.* showed a patency rate of 86.3 per cent after one year, 81.9 per cent after two years, and 79.5 per cent after three years post femoro-popliteal bypass surgery.[12] In comparing patency rates, it is important to note the differences between the angioplasty patient population and the patients undergoing surgical bypass grafting, for the angioplasty patients are generally in worse medical condition and are often refractory to surgery.

The role of antiplatelet, anticoagulant, or fibrinolytic therapy in angioplasty is yet to be clearly defined. Horváth recently showed increased patency rates in total femoral occlusions of greater than 10 cm with the use of an oral fibrinolytic agent, SP-54 (sodium pentosanpolysulphate).[13] Technically the angiographic and angioplasty catheters are undergoing marked improvement; this will allow more lesions to be crossed in the future.

We conclude that PTA is an effective alternative in the management of many patients with ischemic superficial femoral artery disease. It appears that long term patency rates are nearly comparable to those achieved surgically and will continue to improve, although further followup is needed. PTA is a cost-effective procedure, as shown in the cost analysis by Vawter in *Table III*.[14] Patients who undergo PTA of the superficial femoral artery, instead of a totally uncomplicated femoro-popliteal bypass grafting, were able to cut 46 per cent of hospital costs and 65 per cent of the hospital stay. The patients are able to recuperate in a matter of days and return to their original work. PTA can also be used in conjunction with surgical therapy to improve proximal or distal flow to a bypass graft. The role of PTA is especially important in the medically complex and poor surgical risk population. In the future, we feel that PTA will have an increasingly important role in the management of ischemic vascular disease.

Acknowledgement

Barbara Ward, R.N., provided special assistance in the preparation of this paper.

TABLE III
COMPARISON BETWEEN SUPERFICIAL FEMORAL PTA AND TOTALLY UNCOMPLICATED FEMORAL-POPLITEAL BYPASS

	Average Hospital Costs	*Average Hospital Stay (Days)*
Femoral-popliteal bypass, totally uncomplicated	$3,356	10.5
Superficial femoral PTA	$1,803	3.6
Savings	$1,553 (46%)	6.9 (65%)

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Prostatic Carcinoma

Report of a Case Presenting With Dysphagia Due to Eaton-Lambert Syndrome

GANJUR H. GURUPRAKASH, M.D.,* *Leavenworth;*
IFTEKHAR AHMED, M.D.,† *Kansas City, Missouri, and*
ALI SHADCHEHR, M.D.,‡ *Leavenworth*

Carcinoma of the prostate is associated with many medical and neurologic complications. These include disseminated intravascular coagulation,[1] nonbacterial thrombotic endocarditis,[2] the syndrome of inappropriate secretion of antidiuretic hormone,[3] and ectopic secretion of ACTH.[4] Common neurologic complications are metastases to the spine. Complications not related to metastasis have also been reported.[5, 6] Reported here is a case of carcinoma of the prostate associated with Eaton-Lambert syndrome (ELS).

Case Report

A 59-year-old man had difficulty in swallowing for one month prior to admission. The difficulty was initially only with liquids, but soon progressed to dysphagia with both solids and liquids. He lost 7 kg in one month. He was seen by an otolaryngologist about two weeks prior to admission. Results of a barium swallow were reported as normal, and an indirect laryngoscopy showed puddling of mucus in both pyriform sinuses. No paralysis of the vocal cords or tumor was seen. The patient had an obvious mass involving the right clavicle which was first noticed about six months earlier and was growing slowly. He had no pain, but complained of limitation of movement of the right shoulder. Review of symptoms revealed that he had nocturia, hesitancy of urination, and dribbling of urine for the past year.

Physical examination showed the patient to be emaciated. There was a hard mass measuring 6 x 4 cm involving the lateral end of the right clavicle. A bruit was heard over the swelling. There was another mass involving the right scapula. All movements of the shoulder joint were limited. Examination of the heart, lungs, and abdomen yielded normal results. Rectal examination revealed an enlarged prostate which was firm. Neurologic examination did not reveal any skeletal muscle weakness; deep tendon reflexes were normal and there was no sensory deficit. As oral intake was poor, a nasogastric tube was inserted for feeding.

Eaton-Lambert syndrome is most often associated with carcinoma of the bronchus, and has been described with varying incidence with other carcinomas. It is believed, however, that the case reported here is the first documented case reported in the English literature in association with carcinoma of the prostate.

Hemoglobin was 12.4 gm/100 ml; hematocrit, 36.4%; BUN, 23.5 mg/dl; creatinine, 1.6 mg/dl; and electrolytes were normal. SGOT was 13 I U/L; LDH, 205 I U/L; SGPT, 4 I U/L; alkaline phosphatase, 780 I U/L; calcium, 9.2 mg/dl; and acid phosphatase, 180 I U/L.

Chest x-ray revealed an osteoblastic process involving the right clavicle *(Figure 1)*. A small amount of barium aspirated during an upper gastrointestinal study was seen in both lower lobes.

Computerized tomography of the chest showed an extensive destructive lesion involving the right scapula. A technetium diphosphonate bone scan revealed increased tracer concentration, compatible with metastatic disease, in multiple areas including the right clavicle. A biopsy of the prostate and the lesion in the clavicle revealed adenocarcinoma. The specimen from the clavicle was positive for acid phosphatase stain, indicating that it was metastatic from the prostate.

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† Clinical Assistant Professor of Neurology, University of Missouri; and Director, Neurodiagnostic Laboratory, St. Mary's Hospital, Kansas City, Missouri.
‡ Veterans Administration Medical Center, Leavenworth.
This paper was presented at the American College of Physicians Regional Meeting, Topeka, February 20, 1981.

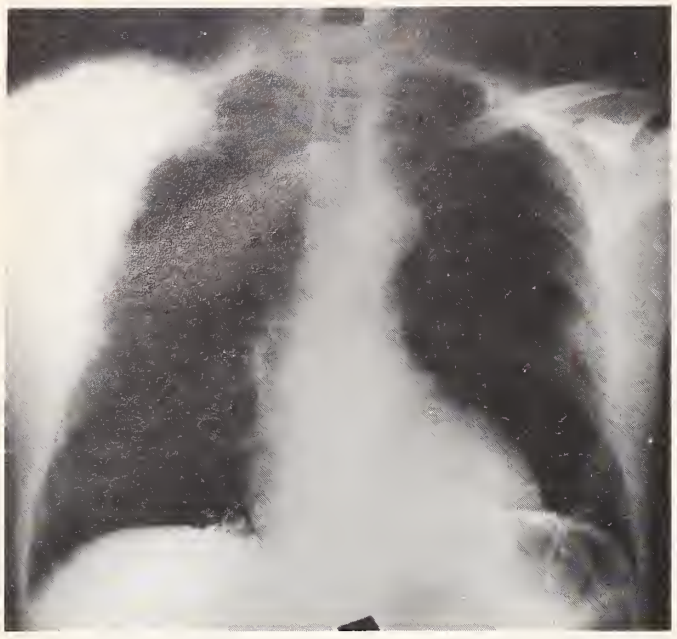


Figure 1. Chest x-ray showing osteoblastic lesion in the right clavicle and specks of barium in both lower lobes.

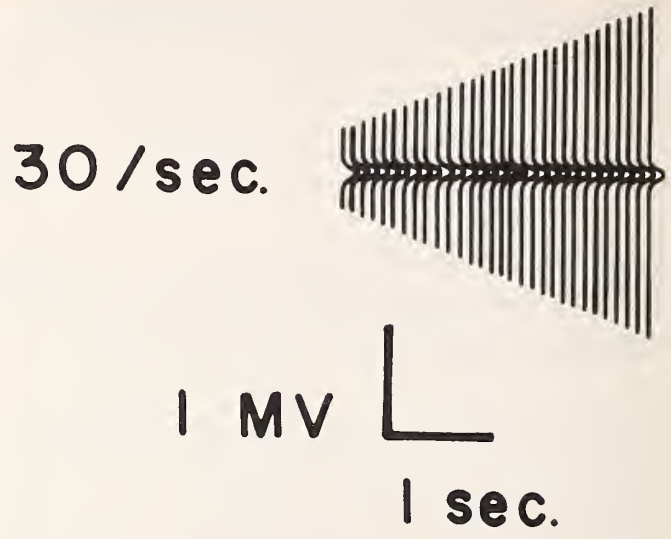


Figure 2. Electrodiagnostic study showing facilitation with supramaximal stimulation.

Electrodiagnostic studies — Mean duration of motor unit potentials of left deltoid and biceps were shortened 35 per cent. Motor nerve conduction velocities were normal in the left median and ulnar nerves. Repetitive supramaximal stimulation was performed and recorded from the deltoid muscle with surface electrodes. The amplitude of the evoked potential was 0.8 mv on 30/sec stimulation. Facilitation occurred to >300 per cent consistent with the diagnosis of Eaton-Lambert syndrome *(Figure 2)*. Esophageal motility studies showed normal upper and lower esophageal sphincters and extremely weak peristaltic waves in the body of the esophagus *(Figure 3)*.

The patient was treated with guanidine hydrochloride, 1500 mg/day. About six days later, the patient noticed improvement in dysphagia and the nasogastric tube was discontinued. A repeat esophageal motility study showed marked improvement in the amplitude of the peristaltic waves. A repeat electrodiagnostic study showed 150 per cent. Carcinoma of the prostate was treated with diethylstilbestrol diphosphate and bilateral orchiectomy. Guanidine hydrochloride was discontinued preoperatively and was not restarted as he remained asymptomatic.

Discussion

Our patient had Eaton-Lambert syndrome (ELS) associated with carcinoma of the prostate. The electrodiagnostic studies showed improvement in the amplitude of the evoked potential of 300 per cent with repetitive supramaximal stimulation. Guanidine, a specific drug for ELS, improved his dysphagia, and there was a demonstrable improvement in his esophageal motility. All these findings were consistent with the diagnosis of ELS.

Carcinoma of the prostate is common in the elderly and may be associated with other malignancies. Thyroid scan, intravenous pyelography, and complete gastrointestinal studies failed to reveal any lesions suspicious of malignancy. Dysphagia markedly improved after treatment of the primary tumor, indicating an association between ELS and the carcinoma of the prostate.

ELS is generally associated with carcinoma of the bronchus, although it has been described in patients

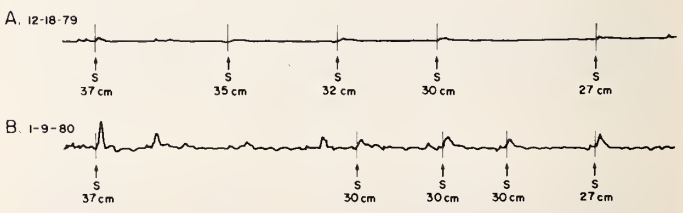


Figure 3. Esophageal motility study. A. Before therapy. Low amplitude of the peristaltic waves (1-2 mm Hg). B. During guanidine therapy. Marked increase in the amplitude of the peristaltic waves (6-8 mm Hg). S = Swallowing.

with carcinoma of the breast, stomach, and rectum. The clinical picture is characterized by muscular fatigability and weakness. Muscles of the pelvic girdle and thigh are commonly involved first, causing difficulty in walking or climbing stairs. As the disease progresses, symptoms of autonomic dysfunction may predominate. In contrast to classic myasthenia gravis, involvement of the ocular and bulbar muscles is rare. Electrodiagnostic studies yield characteristic results, and help to differentiate ELS from myasthenia gravis. The defect in the neuromuscular transmission is believed to be a decrease in the release of acetylcholine at the endplate. Guanidine, which promotes the release of acetylcholine, is effective in the management of these cases.

In the 1974 edition of *Disorders of Voluntary Muscles,* carcinoma of the prostate is listed as one of the diseases associated with ELS,[7] but no references are listed. Brain & Henson reviewed 42 cases of carcinomatous neuromyopathies; 32 occurred in patients with carcinoma of the lungs, and of the remaining 10 patients, two had carcinoma of the prostate. There were 13 patients with diplopia, ptosis, and bulbar paresis. Myasthenia was an occasional feature in these patients as some responded to neostigmine. The majority of these 13 patients had cancer of the lung, but several had rectal and prostatic growth. As patients with ELS do not respond to neostigmine, it is probable that these patients had myasthenia gravis. Whether the patients with carcinoma of the prostate responded to neostigmine is not reported. No electrodiagnostic studies were done.

Newman *et al.* reported 16 cases of carcinomatous neuropathies and myopathies; four patients had cancer of the prostate, but none had ELS.[6] We believe that this is the first documented case reported in the English literature of ELS in association with carcinoma of the prostate.

Another interesting feature of our patient is the presence of metastasis to the clavicle and scapula which simulated a primary bone tumor. There are reports of osteoblastic metastasis from carcinoma of the prostate simulating primary osteogenic sarcoma. There are also reports of steogenic sarcoma occurring in a metastatic lesion from carcinoma of the prostate, giving rise to a speculation that osteoblastic metastatic process and osteogenic sarcoma may have a cause and effect relationship.[8]

Dysphagia was the only symptom of ELS present in our patient. This syndrome should be considered in the differential diagnosis of dysphagia in a patient with any malignant disease.

Acknowledgement

George Trimble, M.D., Director of Medical Education, Saint Mary's Hospital, Kansas City, Missouri, reviewed this manuscript.

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Physical Examinations

In the September 1982 KMS *Newsletter,* guidelines for physical examinations were noted from the recommendations of the AMA Council on Scientific Affairs report on Medical Evaluations of Healthy Persons. The following guidelines are in keeping with the recommended standards of the American Academy of Pediatrics. However, it should be stressed that the Academy recommendations are flexible and emphasize the necessity for meeting the needs of each child. Additional visits may become necessary if circumstances suggest variation from normal.

Category	*Frequency of Examination*
Newborn	Two to four weeks after birth
1st year of life	Four or five more times
Through age 21 years	One to two year intervals
Until age 40 years	Five year intervals
After age 40 years	Periodic intervals of one to three years depending on individual

Upper Airway Obstruction

Respiratory Insufficiency in COPD

HELMY H. TAWADROS, M.D.,* *Topeka*

EARLY DIAGNOSIS of upper air flow obstruction (UAO) is often difficult. The condition may be unrecognized in patients with chronic obstructive pulmonary disease (COPD). Obstructive lesions of the larynx and trachea are rarely considered as a cause of dyspnea and wheezing in an adult, and the symptoms are often attributed to asthma, chronic bronchitis, emphysema, or recurrent pulmonary infections. Since treatment of major airway obstruction is quite specific and often surgical, recognition of the strategic sites of the causative lesions is essential.

Documented here are four instructive cases in which the history and physical findings were suggestive of UAO. The flow-volume loops (F-V) were helpful in definitive endoscopic or roentgenographic confirmation of the site of obstruction.

Case One: A 65-year-old male was admitted to the hospital because of severe dyspnea. He had a prolonged history of COPD with repeated admissions for acute exacerbations, but no history of allergy or occupational exposures. He had recently developed an upper respiratory infection which failed to clear in spite of conventional treatment as an outpatient. Chronic pulmonary symptoms worsened with increased productive cough and purulent sputum, wheezing, and increased shortness of breath. There was no hemoptysis or chest pain, but he had a mild fever with no rigors. During the few days prior to admission, the patient experienced increased hoarseness and paroxysms of violent croupy cough, accompanied by stridor and choking sensations, especially during the night. A syncopal episode following a violent spasm of cough was followed by a short-lived seizure. Previous pulmonary function tests showed moderately severe COPD. Forced vital capacity (FVC) was 62 per cent of predicted, and forced expiratory volume in one second (FEV_1) was 33 per cent of predicted. The ratio of forced expiratory flow (FEF) and forced mid-expiratory flow (FMF) was moderate at 40 per cent of predicted; maximum breathing capacity (MBC) was 45 per cent of predicted. Arterial blood gases (room air) were: PO_2, 58.4; pCO_2, 46.3; pH, 7.39; HCO_3, 28.5; and SaO_2, 89%. The flow volume loop *(Figure 1a)* showed expiratory flow limitation more marked at mid and low lung volumes consistent with COPD.

At admission, the patient was in moderate respiratory distress and had cyanosis of lips and nail beds.

Upper airway obstruction is often unrecognized, especially in patients with preexisting chronic obstructive pulmonary disease. Correct diagnosis is essential because treatment is quite specific. Once diagnosed and properly treated, gratifying results with relief of most symptoms are the rule.

Blood pressure was 180/100 mmHg; temperature, 38.1C; pulse, 120/min; and respirations 23/min. There was increased anteroposterior (AP) diameter and decreased breath sounds throughout both lung fields. Other clinical symptoms included inspiratory and expiratory tracheal wheezing and a few rales in bases on the left. Results of the rest of the examination were unremarkable. Arterial blood gases were: PO_2, 45; pCO_2, 77.5; pH, 7.32; HCO_3, 43; and SaO_2, 80%. Chest roentgenogram is shown in *Figure 2*. The flow volume loop *(Figure 1b)* showed the new development of a plateau of air flow in both inspiration and expiration, and was consistent with fixed upper airway obstruction. Laryngoscopy under topical anesthesia revealed an extensive supraglottic mass lesion that reduced the distance between the vocal cords to a slit of 3 x 1 mm. Biopsy of the lesion showed invasive squamous cell carcinoma of the larynx. Emergency tracheostomy was performed with prompt relief of obstruction and progressive improvement of respiratory status *(Figure 2b)*. The patient was later referred for definitive treatment by radiotherapy for laryngeal carcinoma.

Case Two: A 59-year-old white male transferred from another hospital with a possible diagnosis of metastatic carcinoma of the larynx to the supraclavicular lymph nodes (node biopsy showed poorly

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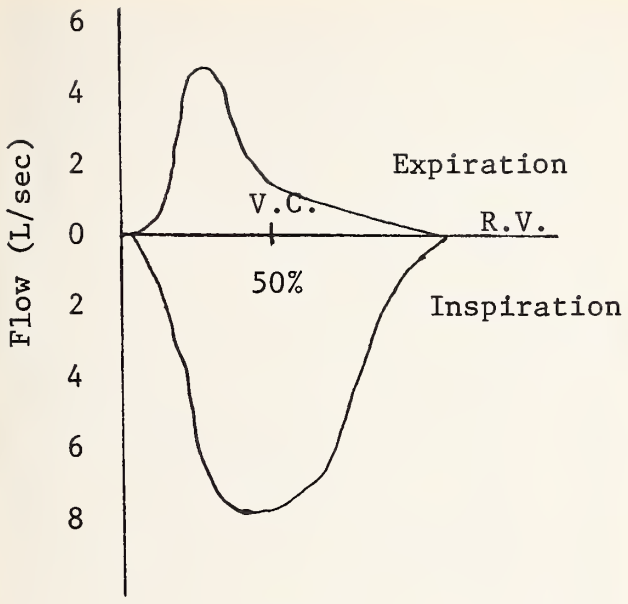


Figure 1-A

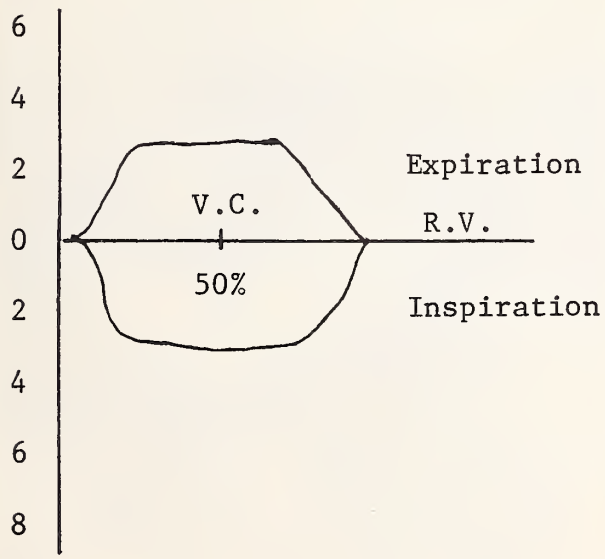


Figure 1-B

Figure 1. (Case One) The F-V loop plotting the lung volumes on the horizontal axis and the flow on the vertical axis with maximal expiratory and inspiratory movements. 1a shows moderately reduced expiratory flow more marked at mid and low lung volume. Inspiratory flow is nearly normal. 1b shows limitation of flow in both inspiration and expiration with a plateau in both phases, consistent with fixed obstruction.



Figure 2a. (Case One) Marked hyperinflation of both lung fields, more marked in the bases.



Figure 2b. (Case One) After relief of laryngeal obstruction by tracheostomy.



Figure 3. (Case Two) Chest x-ray shows a left hilar mass lesion with multiple bilateral modular densities consistent with multiple metastases.

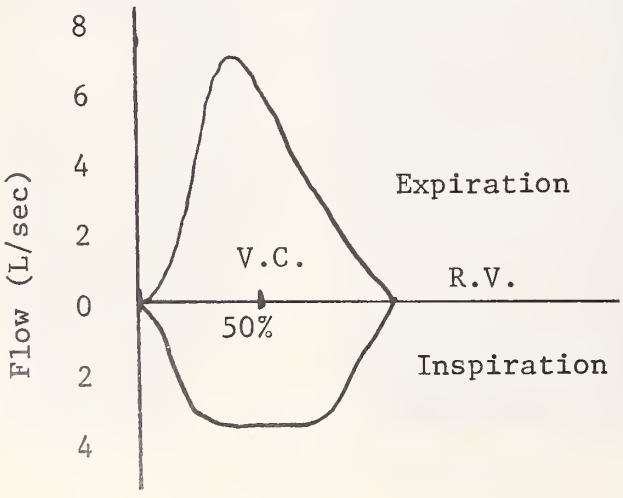


Figure 4. (Case Two) F-V loop shows limitation of inspiratory flow with plateau, while expiration is nearly normal. This is consistent with extrathoracic variable obstruction (vocal cord paralysis).

differentiated squamous cell carcinoma) and metastasis to the lungs and mediastinum. The patient was in poor general condition with obvious hoarseness and dysphasia, terminally ill, and was unable to give any history.

Clinically, he was in moderate respiratory distress with bilateral palpable cervical lymph nodes, mild hyperinflation, poor intensity of breath sounds, and a few rales in both bases. Arterial blood gases were: PO_2, 46; pCO_2, 28; pH, 7.48; HCO_3, 28; and SaO_2, 76%. Chest x-ray was as shown in *Figure 3*. The F-V loop *(Figure 4)* showed flow limitation with a plateau in the inspiratory phase consistent with variable extrathoracic obstruction. Endoscopic studies showed left vocal cord paralysis, but no laryngeal masses were noted. Fiberoptic bronchoscopy showed an endobrachia mass lesion of the left main bronchus, and biopsy showed poorly differentiated squamous cell carcinoma. The patient had a rapid



Figure 5. (Case Two) The gross left lung specimen showing massive tumor at the level of the left main bronchus involving the left recurrent laryngeal nerve.



Figure 6. (Case Two) The gross microscopic specimen of the larynx at autopsy showed no lesion as was predicted by the F-V loop and confirmed by laryngoscopy.

downhill course, and died a few days later. Autopsy findings showed a large left bronchogenic carcinoma involving the left recurrent laryngeal nerve with widespread metastases *(Figure 5)*. No tumor of the larynx was identified *(Figure 6)*. The ante-mortem findings with the F-V loop and later endoscopy avoided unnecessary tracheostomy for this terminally ill patient.

Case Three: A 54-year-old white male with an 18-year history of multiple sclerosis had experienced progressive respiratory difficulties with frequent pulmonary infections dating from the early 70s. He had multiple episodes of respiratory failure and had undergone tracheostomy in 1971 and 1973; on both occasions the tracheostomy continued for a period of three to four months before it was closed. Pulmonary status was relatively stabilized in 1974. In the fall of that year he developed respiratory failure which necessitated endotracheal intubation. When he was seen later as an outpatient, his pulmonary function showed moderate restrictive disease and slightly reduced expiratory flow rates *(Figure 7a)*. Three months later he began to complain of increasing dyspnea and wheezing and that his breathing became more noisy when he was lying down. Clinically, there was poor expansion of the chest, a few rales in the left base, and wheezing centrally over the trachea. No evidence of congestive heart failure was noted. Arterial blood gases were: PO_2, 64; pCO_2, 34; pH, 7.41; HCO_3, 25.3; and SaO_2, 89%. The F-V loop *(Figure 7b)* showed a plateau of limited air flow in both phases of inspiration and expiration denoting fixed upper airway obstruction. Tracheal stenosis

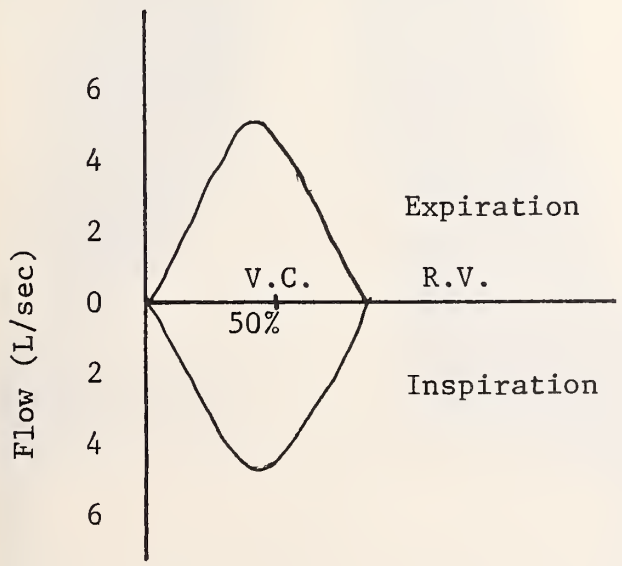


Figure 7a. (Case Three) F-V loop showing relatively small lung volumes consistent with restrictive disease and slightly reduced expiratory flow rates.

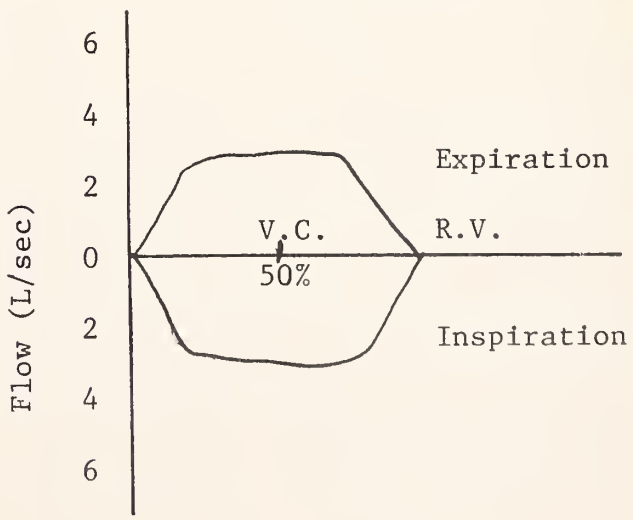


Figure 7b. (Case Three) F-V loop showing limitation of flow in both phases consistent with fixed obstruction.



Figure 8. (Case Three) Chest tomogram showing greater than 50 per cent stenosis of the trachea due to previous tracheostomies.

due to previous tracheostomies was suspected. Tomograms of the trachea *(Figure 8)* confirmed greater than 50 per cent stenosis secondary to the previous tracheostomies.

Case Four: A 56-year-old white male was admitted for treatment of congestive heart failure. He was markedly obese, and had a history of alcohol abuse. He had a 30 pack/year history of cigarette smoking. His primary complaints were increasing shortness of breath during the last six months and leg edema which had developed during the last month. Hypersomnolence had increased during the past two years. The patient's wife found it very difficult to sleep in the same room with him because of loud snoring. She said that at times during sleep he stopped breathing for variable periods; this was followed by twitching, then a change of position, and then loud snoring. The pattern was repeated throughout the night. The patient height was 175 cm; his weight was 125 kg; and he was noted to be plethoric with a short, thick neck *(Figure 9)*. His lungs showed a few rhonchi and bibasilar rales. Cardiovascular examination revealed right ventricular heave, increased P_2, S_4



Figure 9. (Case Four) Patient prior to treatment. Marked obesity (125 kg), plethoric, cor pulmonale, and sleep deprivation.



Figure 10. (Case Four) Chest x-ray showing cardiomegaly with prominent major pulmonary vessels.

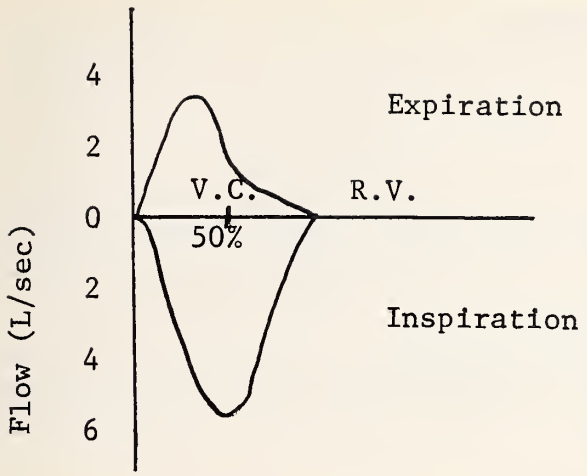


Figure 11. (Case Four) F-V loop showing moderately reduced expiratory air flow at mid and low lung volume.

gallop without murmurs, and jugular vein distension with bilateral leg edema consistent with cor pulmonale and congestive heart failure. Laboratory results included hemaglobin, 18 gm/100 ml; hematocrit, 56%; and red blood count, 6 million. Electrocardiogram showed right axis deviation and right atrial hypertrophy. Arterial blood gas studies showed the following:

Early morning, arterial blood gases, right auricle: PO_2, 52; pCO_2, 60; and pH, 7.34.

Nocturnal, arterial blood gases, right auricle: PO_2, 48; pCO_2, 64; and pH, 7.32.

Hyperventilation arterial blood gases: pO_2, 65; pCO_2, 38; and pH, 7.41.

Chest x-ray showed cardiomegaly with prominent main pulmonary artery *(Figure 10)*. Pulmonary function test results were: FEV_1, 960 ml; FVC, 1634 ml; and $\frac{FEV_1}{FVC} = 59\%$. The F-V loop *(Figure 11)* was consistent with reduced air flow rate in the expiratory phase consistent with moderate COPD. However, the clinical presentation was that of obstructive sleep apnea *(Figure 12)* which was relieved by tracheostomy.

Discussion

Upper airway obstruction is a relatively rare, but often unrecognized, cause of dyspnea and ventilatory failure in the adult. Patients are often treated for long periods of time for diffuse airway obstruction (asthma, chronic bronchitis, or emphysema). However, during the last decade, there has been an increasing awareness among physicians of the frequency of UAO. The increasing incidence is attributed to prolonged use of endotracheal tubes or tracheostomies for mechanical ventilation — especially with the recent trend toward aggressive resuscitation measures — and physical and chemical trauma to the major airways. Laryngotracheal neoplasms and vocal cord paralysis (either postoperative or from other causes) continue to be a significant problem. In addition, the recently identified syndrome of "Obstructive Sleep Apnea" has added a substantial dimension to the diagnostic challenge.

Difficulties in diagnosis stem from the fact that the symptoms are usually non-specific and do not occur until the obstruction is quite advanced. However, the presence of inspiratory stridor (accentuated by forced inspiration, exercise, or hyperventilation),[1] hoarseness, or dyspnea related to

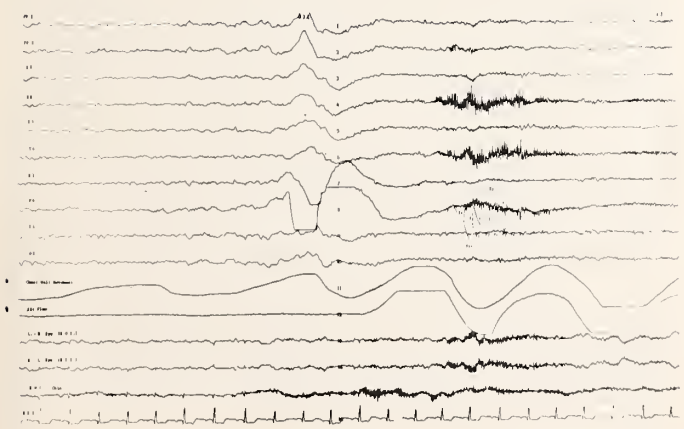


Figure 12a. (Case Four) Sleep study polygraph. EEG monitoring (upper 10 channels), followed by chest wall movement and airflow electrooculogram (EOG), electromyogram (EMG), and electrographic (ECG) monitoring. The absence of air flow in the presence of chest wall movement is indicative of sleep apnea syndrome.

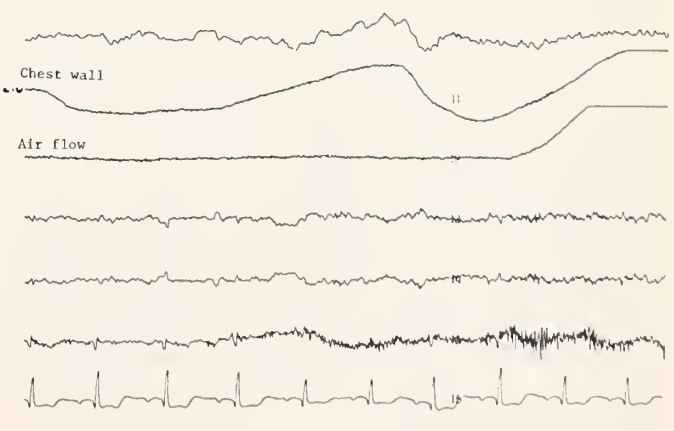


Figure 12b. (Case Four) Close-up showing no air flow in the presence of chest wall movement.

certain body positions are all suggestive of upper airway obstruction.

The use of pulmonary function tests, particularly F-V loops, can be very useful in the diagnosis of UAO. Scanning the literature shows that several reports have dealt with ventilatory aspects of obstructing lesions of major airways. Engstrom,[2] and later Shim and associates,[3] found mid-inspiratory flow to be disproportionately decreased in relation to mid-expiratory flow in patients with upper airway obstruction. Sackner[4] showed that obstructing lesions of the upper airways are associated with normal distribution of ventilation and elevation of airway resistance unresponsive to bronchodilators. Harry Bass,[5] using F-V loops, showed that patients with COPD have limitation of expiratory flow at mid and low lung volumes (effort independent) partially improved by helium, while inspiratory flow is nearly normal. Miller and Hyatt,[6, 7] in their studies of upper airway obstruction, used F-V loops to characterize three major patterns which were completely distinct from that of diffuse airway obstruction (asthma, bronchitis, or emphysema). At present, UAO can be subdivided into four functional groups.[8]

Fixed Obstruction. This includes such conditions as tracheal stenosis and laryngeal tumor. In this type of lesion, the airway diameter does not change with inspiration or expiration. The constant air flow is represented by a plateau in the expiratory and inspiratory curve in the effort dependent portion of the F-V curve. The expiratory/inspiratory flow ratio at 50 per cent of vital capacity (VC) — i.e., mid VC ratio — will be equal to one (as shown in cases one and three).

Variable Extrathoracic Obstruction. Vocal cord paralysis is the most common cause. This will lead to limitation of the flow during inspiration leading to a plateau in that phase (Case Two). The mid VC ratio is greater than one.

Variable Intrathoracic Obstruction. This may result from such conditions as floppy airway or tracheal tumor. This causes limitation of flow in expiration leading to a plateau in that phase. The ratio at 50 per cent VC is less than one.

Sleep-Apnea Syndrome. This is caused by occlusion of the upper airways during sleep. It requires monitoring of electroencephalogram, air flow, respiratory movements, and electrocardiogram. The absence of air flow in the presence of respiratory movements are indicative of obstructive sleep apnea[9, 10] *(Case Four).*

Conclusion

Increased awareness of upper airway obstruction can facilitate early diagnosis through pulmonary function studies utilizing F-V loops. Further definitive studies for localization of the site of the obstruction can be defined by roentgenography (tomography or bronchography) and by endoscopy (laryngoscopy and fiberoptic bronchoscopy). In the clinical setting of sleep apnea, sleep studies should be performed for the definitive diagnosis of the obstructive type. Once diagnosed and properly treated, gratifying results with relief of most of the symptoms is the rule.

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Non-Invasive Carotid Screening

Correlation With Angiography

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and* RICHARD A. AHLSTRAND, M.D., *Wichita

Tests for carotid artery flow which do not subject the patient to risk or discomfort have been investigated in recent years, and these tests have been termed "non-invasive."[1] Because of the potential for surgery with carotid bifurcation disease, identifying hemodynamically significant plaques in patients could prevent a number of strokes. With definite symptoms of transient cerebral ischemia, arteriography can be employed in time for surgical intervention. But many strokes occur without warning; thus we need a diagnostic approach other than catheter angiography for the large number of adults with symptoms, signs, or risk factors suspicious for cerebral ischemia.

Methods

At Wesley Medical Center, we have employed three tests: the oculopneumoplethysmograph or Gee method (OPG), direct Doppler of the carotid artery with audio and spectral analysis, and real time imaging with pulse Doppler flow sampling (duplex scanning).

The OPG test, developed by William Gee,[2] determines the pressure in each ophthalmic artery and compares the arrival time of the pulse to right and left eyes. A soft plastic cup is placed over each anesthetized eye and connected to a vacuum pump. When activated, a predetermined pressure is rapidly obtained which immediately causes bilateral interruption of ophthalmic artery flow followed by a rapid, constant fall of the vacuum to zero. Sensors in the eye cups are activated as the pulses return, and pulsations are graphed on a strip chart recorder. Pressures and comparisons between eyes are read directly from the recording.

Direct Doppler examination of the carotid arteries is done by employing a continuous wave probe along the course of the common, internal, and external carotid arteries.[3] The pitch of the audio signal rises at stenotic areas. Also, the signal can be displayed as a spectrum of all velocities present, again, the higher frequencies being found at areas of constriction.

Three non-invasive tests were used as a battery to screen patients with symptoms suggesting possible cerebral ischemia. In 76 patients who ultimately had angiography, the non-invasive battery was 88 per cent correct in predicting 50 per cent or greater stenosis of one internal carotid artery, and 91 per cent correct in predicting negative or insignificant disease in the carotid arteries.

Real time imaging gives an anatomic picture of the vessel under the probe.[4] The position can be rapidly changed in order to identify the common carotid, the bulb, and the internal and external branches. These can usually be seen for 2-3 cm above the bifurcation, depending on the position of the jaw. Also, flow within the lumen being visualized can be simultaneously "listened to" by means of Doppler flow sampling of a small discrete volume within the vessel.[5]

Results

Results of all tests are shown in *Tables I* and *II*.

Between January 1981 and February 1982, 375 patients underwent non-invasive carotid screening. Seventy-six patients subsequently had angiography; 34 patients had negative or insignificant disease and 42 patients showed stenosis of 50 per cent or greater in at least one internal carotid artery.

The OPG was positive in 56 per cent of the 42 true positive cases and was positive erroneously only one time in 34 cases.

The Doppler was positive in 56 per cent of true positives and never positive (frequency above 10,000 Hz) in 34 true negative cases.

Duplex scanning was positive in 80 per cent of true positive cases and was positive erroneously in two of 34 negative cases.

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TABLE I
42 POSITIVE ARTERIOGRAMS*

	True Positive		*False Negative*	
	#	%	#	%
OPG	23	56	19	44
Direct Doppler	23	56	19	44
Duplex scan	34	80	8	20
Combined battery	37	88	5	12

* Fifty per cent or greater stenosis in one or more internal carotid arteries, or ulcerated, non-stenotic plaque in an internal carotid artery.

If any one test in the battery was positive, this was considered hemodynamically significant carotid stenosis.

Utilizing these criteria, the non-invasive battery was 88 per cent accurate in predicting hemodynamically significant carotid stenosis and was 91 per cent accurate in predicting negative or insignificant carotid artery disease.

As in many studies, there were relatively more errors in the early months because of observer inexperience and equipment malfunction. No patients were excluded from analysis.

Discussion

Non-invasive tests have studied the ophthalmic arteries and their branches, the facial arteries, and the carotid arteries themselves. Plethysmography and ultrasound have usually been the basic types of test, although thermography and other modalities have been employed. No single test has proven to be superior, and most workers use two or more tests in their screening programs.

TABLE II
34 NEGATIVE ARTERIOGRAMS

	False Positive		*True Negative*	
	#	%	#	%
OPG	1	3	33	97
Direct Doppler	0	0	34	100
Duplex scan	2	6	32	94
Combined battery	3	9	31	91

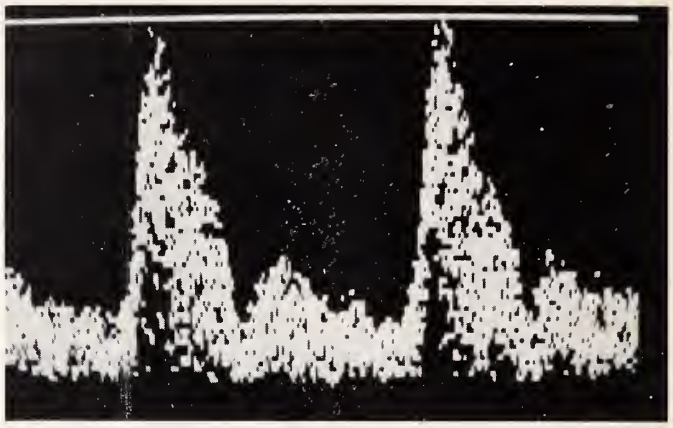


Figure 1. Normal spectral display — Doppler from common carotid artery. Peak frequency — 3,920.

The OPG has been quite useful with high-grade stenosis or occlusion. This test is usually normal with milder disease. For severe unilateral stenosis, a delay in the return of the pulse will occur on the affected side. With bilateral disease, abnormalities of pressure may be equal and more difficult to recognize. With less than 60 per cent stenosis, or whenever collateral flow has had time to be well established, the test will be normal.

Direct Doppler has proven to be most useful with 50-90 per cent stenosis. Smaller plaques which do not reduce the cross-sectional area significantly do not alter the velocity of flow (hence, a normal fre-

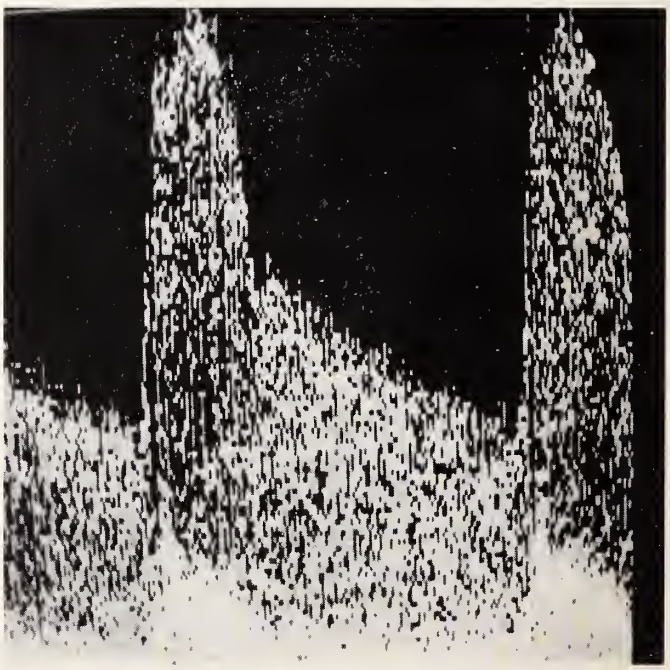


Figure 2. Ninety per cent stenosis of an internal carotid artery. Spectral display of Doppler signal showing turbulent flow (multiple lower frequencies) as well as a higher peak frequency — 20,320.

quency). Also, occlusion will result in no Doppler signal. *Figures 1* and *2* show Doppler spectral displays of two patients.

Duplex scanning has been very accurate in normal vessels and in mild to moderate stenosis. With severe disease the vessels become smaller, irregular, and more difficult to see.

The entire battery of three tests can be performed in 45-60 minutes by experienced vascular sonographers. The test results are reviewed by the radiologist, who usually repeats some of the duplex scans to better understand the relationships of vessels, plaques, and velocity of flow. If, on the basis of one or more tests, it is felt that hemodynamically significant disease is present in an internal carotid artery, an arteriogram is recommended. Hemodynamically significant disease is defined as 50 per cent or greater stenosis with the diameter of vessel at plaque as a percentage of normal diameter of the affected vessel.

One important fact discovered is the importance of the OPG whenever there is occlusion or severe stenosis. The duplex scan becomes less reliable as the smaller irregular vessels are more difficult to image, and Doppler will not detect any high velocities if the flow is reduced to a trickle or even less. Here, the OPG merits its inclusion in the battery as the ophthalmic artery pressure differences will alert the examiner to the true reason for the less than ideal images of the affected arteries.

Ulcerated, non-stenotic plaques are a source of strokes which plague the sonographer. Usually they cannot be seen unless the scanning plane is parallel with the ulcer and it is large enough to be resolved. Of course, small ulcers are difficult to detect angiographically as well.

Soft thrombus may have the same appearance as a column of blood on real time. Hence, flow sampling should be combined with imaging, although this is a very difficult technical problem. Current duplex scanners have indeed achieved amazing results but improvements still need to be made. A real time image and duplex scans are shown in *Figure 3*.

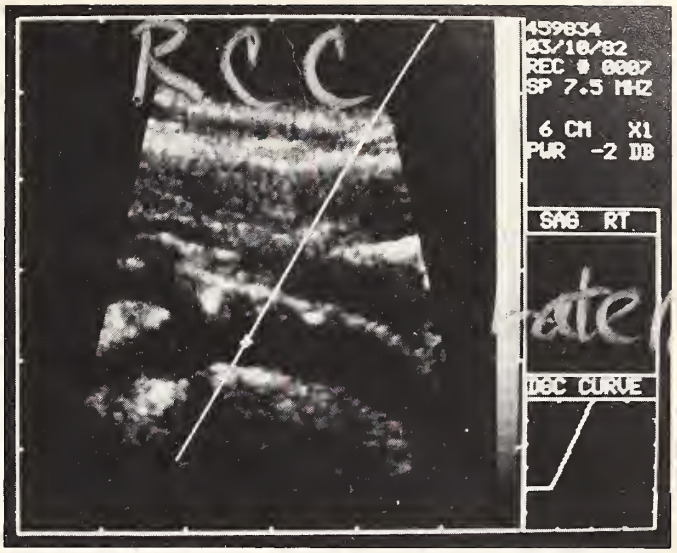


Figure 3b. "Real time" image showing common carotid, bulb, and proximal internal and external carotid arteries. Small, dense plaque at bulb.

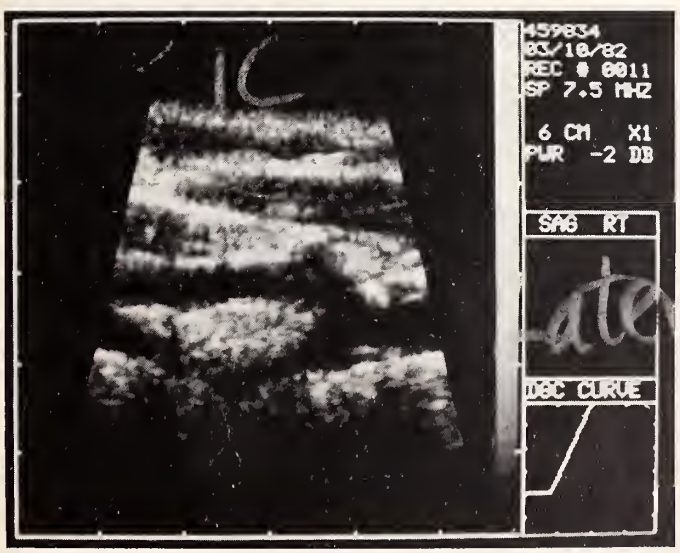


Figure 3a. "Real time" image — carotid bulb and internal carotid artery showing probable large ulcer proximal internal carotid artery in a 67-year-old woman with massive stroke.

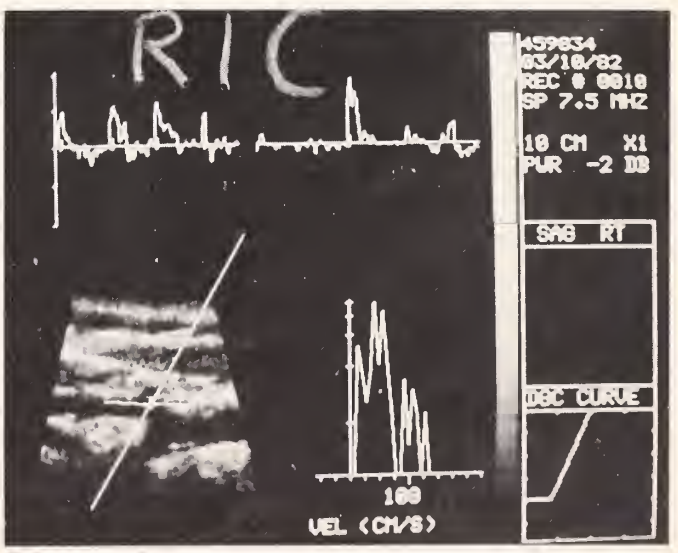


Figure 3c. Duplex scan — common carotid, bulb, internal carotid. Flow sample — proximal internal carotid artery. Velocity of flow in cm/sec given at lower right.

The future role of digital subtraction techniques in carotid occlusive disease will have an impact on non-invasive carotid testing and probably on conventional catheter angiography. At present most centers employ angiography prior to surgery despite accurate non-invasive tests. Other lesions at the arch, intracranial disease, and more definition of the carotid lesion are the reasons for this philosophy. Also, surgeons tend to favor a familiar anatomic image to relate to in performing this kind of surgery.

The role of non-invasive carotid testing has been undergoing continuous modification throughout its history. Its reliability combined with its lack of risk or discomfort will insure its continued place in the prevention of stroke.

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Tax Equity Act

(Continued from page 604)

- provide indicated post-anesthesia care.

In situations where the anesthetist is actually employed by the anesthesiologist, reimbursement under Part B would be authorized at a rate of one "time unit" for each 15-minute interval. Where the anesthetist is not employed by the anesthesiologist, reimbursement under Part B would be authorized at a rate of one "time unit" for each 30-minute interval.

The proposed rule would also apply the RCE as a cap on total reimbursement to a physician for anesthesiology services.

Radiology Services

Part B reimbursement for radiology services by a physician to an individual patient would be recognized if the services are "identifiable, direct, and discrete diagnostic or therapeutic services to an individual patient, such as interpretation of x-ray plates, angiograms, myelograms, pyelograms, or ultrasound procedures." Where radiology services are furnished in a provider setting, Part B charges would not be recognized beyond 40 per cent of the prevailing charge for similar services provided in non-provider settings, to reflect the lack of overhead and equipment costs to the physician for the service provided in the institution.

The proposed rule would also apply the RCE as a cap on total reimbursement to a physician for radiology services.

Physician Laboratory Services

These regulations would authorize Part B reimbursement for anatomical pathology services, consultative pathology services, or services performed by a physician in personal administration of test devices, isotopes, or other materials to an individual patient. In addition, consultative services would be recognized as a Part B reimbursable service only if specified conditions are met. To qualify, the service must:

- be requested by the patient's attending physician;
- relate to a test result *that lies outside the normal range established by the laboratory;*
- result in a written narrative report for inclusion in the patient's medical record; and
- require the exercise of medical judgment by the consultant physician.

The proposed rule would also apply the RCE as a cap on total reimbursement for physician laboratory services.

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Bilateral Wilms' Tumor

A Case Report and Review of the Literature

TRIBHAWAN S. VATS, M.D.,* *and* **JAMES SEEMAN,** *Kansas City, Kansas*

WILMS' TUMOR (nephroblastoma) is the second most frequent solid tumor and the most frequent intra-abdominal tumor in children. Approximately 450 cases of Wilms' tumor are diagnosed each year.[1] The interest in and the importance of finding the most appropriate treatment modalities for this disease have led to extensive clinical research. The formation of the National Wilms' Tumor Study (NWTS) groups was an outgrowth of the need for coordinated study and evaluation of diagnostic, staging, and treatment techniques for Wilms' tumor. These studies have provided valuable information, and the success rate for treatment of Wilms' tumor has improved gradually during the past several years. However, the NWTS and most other independent studies concentrate their efforts on disease stages I through IV. Bilateral Wilms' tumor accounts for 5-12 per cent of all Wilms' tumors.[2] One common denominator for almost all therapeutic regimens for both unilateral and bilateral disease is surgery in the form of nephrectomy and in bilateral cases, partial resection of the contralateral kidney. Successful treatment without surgery has occurred to varying degrees. In this report, a case of bilateral, non-resectable Wilms' tumor is presented and data on other series is reviewed.

Case Report

A 20-month-old male was first seen at 7 months of age at the University of Kansas Medical Center for the evaluation of bilateral renal masses. These were noted initially during the infant's routine 6-month well-baby check. Subsequently, plain films of the abdomen, an abdominal sonogram, and an intravenous pyelogram demonstrated large bilateral masses located centrally in the kidneys *(Figure 1)*. There was marked enlargement of both kidneys and distortion of the collecting systems.

On admission to KUMC (December 9, 1980), the child appeared quite normal; he was active, alert, and well-developed for his age. Results of his examination were completely normal except for a palpable mass in the left flank which was described as firm, irregular, and located 3-4 cm below the left costal margin. The right mass was not palpable. All laboratory values were within normal limits, and there was no indication of decreased renal function.

Wilms' tumor is the second most common solid tumor seen in children; approximately 450 cases are diagnosed each year. Bilateral tumors account for 5-12 per cent of all cases. In spite of the bilateral presentation, the survival rate is fairly good. Surgery at the time of presentation or subsequently is the most important prognostic factor related to survival. Chemotherapy and radiation therapy may help to maintain a disease-free survival.

Laparotomy, performed December 12, revealed bilateral tumors. These tumors were quite large measuring 5 cm in diameter on the right and 6 cm on the left. The tumors were centrally located and could not be resected. There was no other organ involvement, no evidence of localized spread, and no gross lymph node involvement. Biopsies of both masses and the draining lymph nodes were obtained.

The biopsies indicated a well-differentiated nephroblastoma with densely packed tubular structures without intervening stromal or sarcomatous elements. The lymph node was interpreted as reactive without evidence of malignancy.

To rule out metastatic disease, computerized tomography (CT) of the chest, abdomen, pelvis, and head was performed as well as a liver-spleen scan. All studies were negative for metastatic disease.

Since surgical resection was not feasible, it was decided to be very aggressive with this patient's treatment. Therefore both chemotherapy and radiation therapy were initiated. The total radiation dose was 1500 rads to each kidney at the rate of 180 rads/day. The chemotherapy regimen consisted of cycles of vincristine, adriamycin, and actinomycin-

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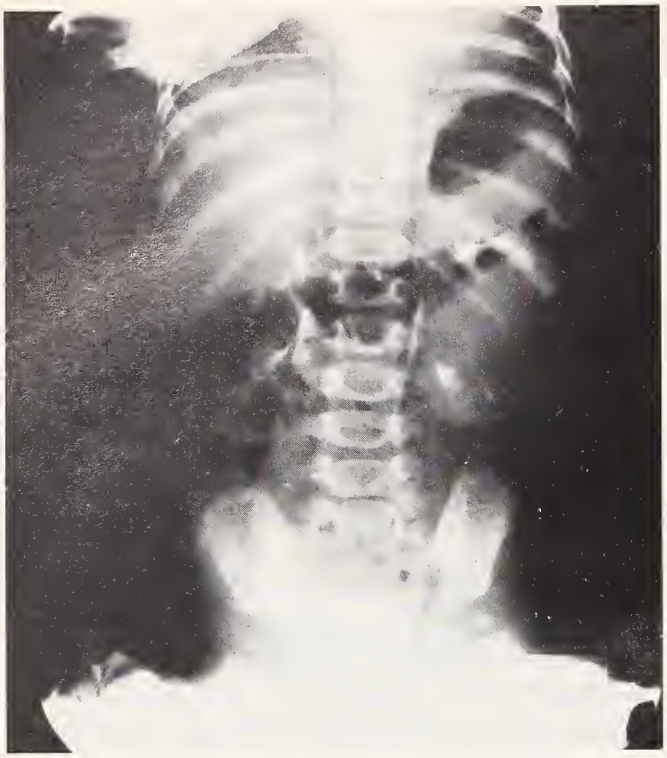


Figure 1. Intravenous pyelogram showing the central location of bilateral Wilms' Tumor.

D as listed in the schedule in *Figure 2*. A Hickman catheter was enplaced for the administration of chemotherapeutic drugs. Treatment was initiated on December 18.

The patient's progress, thus far, has been quite satisfactory. He has had one documented incidence of sepsis and one of fever of undetermined origin. Two months after the initiation of therapy, CT of the chest and abdomen was obtained. The CT indicated a marked decrease in both renal masses with the right mass measuring 2 cm (decreased from 5 cm) and the left mass measuring 2.4 cm (decreased from 6 cm). However, the CT of the chest indicated densities in both the left and right lower lungs which were possible metastases. Chemotherapy was continued and the patient was followed on an out-patient basis.

CT on April 3, 1981 (after four months therapy) revealed a further decrease in the diameter of the renal masses to 1.6 cm and 1.5 cm in the right and left kidneys, respectively. The lung masses, at that time, showed a decrease in size of the left but the right mass remained static. CT on May 22 (after 5.5 months of therapy) showed no further decrease in the size of the kidney masses but they had calcified. The right lung mass again was unchanged but the left was no longer identifiable. Further CTs have continued to show no change in the renal masses and persistence of the right lung nodule. On October 14 (after

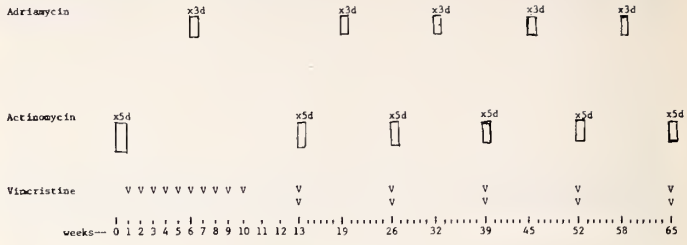


Figure 2. Chemotherapy Schedule: adriamycin = 20 mg/m^2/d; actinomycin = 15 mcg/kg/d; vincristine (V) = 1.5 mg/m^2/wk — all intravenous.

11 months of therapy) the patient was hospitalized for a right thoracotomy and lobectomy. The operation revealed no metastatic nodule but instead demonstrated dense fibrotic material without evidence of malignancy.

The patient currently is continuing with chemotherapy. The most recent CT of the chest and abdomen shows his condition to be stable with no new detectable lesions.

Discussion

Treatment for Wilms' tumor consists primarily of surgerical intervention aimed at complete removal of the tumor, followed by chemotherapy, radiation therapy, or a combination.[1-6] Using these modalities, the survival rate for Wilms' tumor has reached approximately 90 per cent.[7] Surgical resection, either at diagnosis or subsequently, is deemed necessary especially in bilateral tumors.

A review by Malcolm *et al.* of 20 bilateral cases revealed that 19 of the 20 patients had undergone a surgical procedure. There was only one case in which only a biopsy could be done, and that patient died in spite of intensive therapy. This patient had extensive disease at diagnosis, including nodal metastases to the left supraclavicular region.[2] In Bishop's survey of 30 bilateral cases, 11 patients had biopsy only at the time of initial surgery. Six of those children subsequently underwent operations. The five patients who could not undergo a second surgical procedure all survived. The survival rate for the entire study was 73 per cent.[1] These two studies have contradictory survival statistics for patients with bilateral Wilms' tumor in whom surgery was not possible at the time of initial diagnosis or at the time of a subsequent second look operation. The survival rate in these studies could reflect staging as well as other prognostic factors associated with unilateral Wilms' tumor.

The staging of bilateral Wilms' tumor has been left to individual study since the National Wilms'

Tumor Study classified all bilateral disease as stage V, regardless of the patient's presentation and disease characteristics. The prognostic factors to be identified include: age at diagnosis; regional lymph node metastases; penetration and extension beyond the renal capsule; favorable or unfavorable histology; and subsequent treatment given to the patient.[1, 8, 9]

In his study of unilateral Wilms' tumor presentations, Breslow related good correlation with these prognostic factors. The most significant factors for poor prognosis were lymph node metastases and unfavorable histology. Histologic status was the most important factor with a 9 per cent death rate occurring in the favorable histology group and a 60 per cent death rate for the unfavorable histology group. The worst combination in Breslow's study was lymph node involvement coupled with unfavorable histologic findings. Twenty-two of the 26 deaths occurred in patients having this combination.[1]

The refinement of the staging system was published by Cassady *et al.* in his review of 20 bilateral Wilms' tumor cases. This system is based upon the TNM (tumor, node, metastases) system as depicted in *Table I*.[10, 11] Those series demonstrated 100 per cent survival at ten years for the three stage III patients. Those cases were not entirely comparable to the present case because they did have either unilateral nephrectomies or partial resection of the contralateral side. The patient presented in this report was younger, had a favorable histology, and no lymph node metastases or penetration of the renal capsule. The patient is currently disease-free and doing well.

The review suggests that for successful treatment of the Wilms' tumor patient, prognostic factors should be taken into account. In a patient with unfavorable histology or lymph node metastases, aggressive surgery, at diagnosis or subsequently, should be attempted. A combination chemotherapy regimen, possibly using the four drugs suggested by the recent National Wilms' Tumor Study, should be initiated. This drug combination — vincristine, actinomycin-D, cytoxan, and adriamycin — should be initiated to combat the poor response in patients with an unfavorable histology. In patients with a good prognosis, surgery still should be attempted. However, surgery should be avoided if an increased morbidity or chance of mortality in the patient (as demonstrated by different reviews and by the present patient) is possible. A good survival rate is possible even if no surgical resection is done.

TABLE I
TNM SYSTEM, WILMS' TUMOR
(based on Garcia *et al.*[7])

T-1	Tumor encapsulated, less than 550 ml, no capsular collecting system or major vascular invasion, complete surgical removal
T-2	Tumor exceeds one or more of above criteria, but is apparently totally excised
T-3	Tumor is massive, invades other organs, and/or cannot be totally excised
N-0	No Nodes
N-1	Hilar or para-aortic lymph nodes involved
M-0	No non-nodal metastases
M-1	Pulmonary metastases only
M-2	Hepatic, CNS, bony or other metastases

Stage I = $T_1N_0M_0$
Stage II = $T_1N_1M_0$ or $T_2N_1M_0$
Stage III = $T_3N_0M_0$ or $T_3N_1M_0$
Stage IV = any T, any N,M_1
Stage V = any T, any N,M_2

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DES

Effects of In Utero Exposure

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DIETHYLSTILBESTROL (DES), a synthetic nonsteroidal estrogen derived from the stilbene molecule, was first produced by Dodds in 1938. DES was recommended in 1945 as a therapeutic agent to prevent repeated abortions and complications of pregnancy such as premature delivery, intrauterine death, and toxemia. It was believed that these problems were caused by a deficiency in the production of two placental steroids — estrogen and progesterone. DES was thought to stimulate the trophoblastic synthesis of these two hormones. Subsequently, DES was used as therapy for threatened abortions and to reduce perinatal mortality rates when pregnancy was complicated by diabetes mellitus. The recommended dosage of DES was 0.5 mg/day beginning in the sixth or seventh week of pregnancy, escalating at fixed intervals until therapy was completed in the 35th week.

DES has a high degree of potency, is effective when administered orally, and is of low cost to manufacturers; therefore it was quickly accepted in clinical gynecological/obstetrical practice. During the 1940s and 1950s about 1-2 per cent of pregnant women in the United States are believed to have received DES, the number dropping to 0.6-1.0 per cent during the 1960s. It has been estimated that between 10,000 and 16,000 live born females per year between 1960 and 1970 were exposed to DES in utero in the United States.[1] Other studies have estimated that 4-6 million Americans, including mothers and their daughters and sons were exposed to this drug between 1945 and 1955.[2]

The widespread therapeutic use of diethylstilbestrol for conditions associated with pregnancy has created a large population of persons exposed in utero. The identification of after-effects of such exposure has led to development of a comprehensive screening and followup program for women; such a protocol for men has not yet been formulated.

The efficacy of DES was tested in 1953 by Dieckmann and colleagues[3] in a prospective double-blind study of 840 women receiving the recommended dosage and 806 women to whom placebos were administered. These investigators found no benefit among the patients who took the prescribed medication. In 1978, Brackbill and Berendes[4] reviewed the statistics in the paper by Dieckmann *et al.*[3] finding that the original published data clearly showed that DES significantly *increased* abortions, neonatal deaths, and premature births. Unfortunately this

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more recent data was not available in the early 1950s. It is apparent that there is a prolonged latent period in the expression of many of the now recognized side effects of DES in the exposed offspring.

DES is still used today to relieve menopausal symptoms, postpartum breast engorgement, senile vaginitis, kraurosis vulvae, and palliation in carcinoma of the breast and prostate.

The hazards and effects of DES on the developing fetus have been the focus of numerous studies. For purposes of this paper, separate discussions of the effects on female and male offspring will be presented.

Effects on Females

In 1971 Herbst *et al.*[5] of the Massachusetts General Hospital first reported a relationship between the development of clear cell adenocarcinoma of the vagina in young females and their exposure to DES in utero. A Registry of Clear Cell Adenocarcinoma of the Genital Tract in Young Females was subsequently established to centralize all available data on patients born after 1940 who had in utero exposure to maternal exogenously ingested hormones. Information gathered by the Registry plus further investigations have corroborated Herbst's original findings; additionally an increased number of nonneoplastic structural and epithelial abnormalities of the female genital tract were found to be associated with in utero DES exposure. These abnormalities include cervical and vaginal structural malformations — ridges, pseudo-polyps, cockscomb cervix — and ectopic benign glandular epithelium in the vagina (adenosis) and cervix (ectropion). Several studies have estimated these benign abnormalities to occur in up to 95 per cent of exposed individuals.[6]

Vaginal adenosis is defined as the presence of glandular (columnar) epithelium or its mucinous products in the vagina. It is believed to be the postnatal retention of Mullerian-derived glandular tissue within the vagina. Adenosis may appear clinically as a granular red patch or red stippling. Often it may be invisible if the glandular epithelium is replaced by immature squamous epithelium (squamous metaplasia). Since immature squamous epithelium is nonglycogenated, application of iodine (Schiller's or Lugol solution) results in nonstaining of the tissue. Cervical ectropion is characterized by glandular epithelium on the exocervix and appears *clinically* similar to vaginal adenosis. DES-related adenosis is most commonly seen in the anterior wall of the upper portion of the vagina. In approximately one-third of the cases of vaginal adenosis, ciliated cells resembling the lining of the normal Fallopian tube or endometrium are found; such cells are not seen histologically in cervical ectropion.

A third benign lesion found in females with in utero DES exposure is the vaginal or cervical ridge, which consists of a partial or complete band of fibroconnective tissue often covered by glandular epithelium. The ridge may be on the cervix or encircle the upper portion of the vagina. If the ridge completely encircles the cervix, it has been called a pericervical collar; if it is only partial and lies anterior to the cervix, it has been called a hood. The term "pseudopolyp" is used to describe the instance when the ridge is on the portio of the cervix in association with an extensive cervical ectropion giving an appearance of a large endocervical polyp protruding through the os.

Herbst *et al.*[7] have shown in a carefully matched prospective study that 35 per cent of a group of in utero DES exposed women had biopsy proven adenosis compared to only 1 per cent in the control group. Cervical ectropion was found in 85 per cent of the exposed group and 38 per cent of the controls. Transverse cervical and vaginal ridges were present in 22 per cent of the exposed group and in none of the controls. A relationship between these benign lesions and the dosage of DES could not be established in this and other studies. The frequency of these lesions was found to increase in an almost linear fashion relating to the point in pregnancy that DES treatment was initiated. If treatment was started prior to the eighth week of gestation, adenosis was found in 73 per cent of patients, and 100 per cent had cervical ectropion. If treatment began after the 17th week of gestation, vaginal adenosis was present in only 7 per cent and cervical ectropion in 70 per cent.

To date there is no one accepted study that documents adenosis as the precursor to clear cell adenocarcinoma. Vaginal adenosis and cervical ectropion have been shown to be histologically present in association with clear cell adenocarcinoma in approximately 90 per cent of documented cases.[8] Statistics on risk of in utero DES exposed females developing this form of cancer are still in collection process in long-term prospective studies. Currently researchers report that probably less than one exposed woman/1000 will develop this cancer in adolescence.[9] The mean latent period between exposure and development of clear cell adenocarcinoma is 18 years, ages ranging from 7-29 years in documented cases. No definite relationship has as yet been established between dosage or duration of DES exposure to the development of this cancer.

Most often patients found to have clear cell cancer

of the vagina present with vaginal bleeding or discharge; however, 15 per cent of reported cases from the Registry have been picked up while asymptomatic by thorough screening methods. In most cases the tumor is confined to the vaginal wall; one-third of the reported cases are predominantly cervical. Size of tumors range from 3-10cm. Most are polypoid or nodular; recently flat or indurated tumors have been identified. Microscopically, the tumor displays solid, papillary, and cystic patterns comprised of clear cells containing abundant glycogen, hobnail cells characterized by a large nucleus with scant cytoplasm protruding into a lumen with surrounding flattened cells. The identical tumor can occur in the endometrium and ovary but in these sites there has never been an association established with in utero DES exposure; these neoplasms occur in older, postmenopausal women.

Nearly one-quarter of the young women with clear cell adenocarcinoma of the vagina and cervix have either died or developed recurrent tumor; this figure is tentative in face of the relatively short followup period of affected women.[10] Factors related to survival include size of tumor, depth of invasion, and presence of lymph node metastasis. Clear cell cancer of the vagina and cervix appears to have a propensity for metastasis to the lungs and supraclavicular lymph nodes. Treatment modality includes hysterectomy, pelvic lymphadenectomy, vaginectomy, and radiation (external or radium needles locally implanted). Wide local excision of tumor has not proven effective. Chemotherapy with multiple drug regimens has been tried with good results in a few cases with general metastasis.[10] Recently the identification of elevated serum activity (cathespin B_1) in tumor patients suggests a potential mode for detection of tumor recurrence in followup of patients.[11]

In the late 1970s, Kaufman *et al.*[12] began to describe several characteristic upper genital tract abnormalities associated with in utero DES exposure, utilizing hysterosalpingography (HSG). Kaufman reviewed HSGs on 66 women selected from the DES screening clinic at Baylor College of Medicine. Radiographic findings included a T-shaped uterus with bulbous cornual extensions arising from the upper end of the uterine cavity in 23 women. The lower two-thirds of the uterine cavity appeared narrowed in many instances. In eight HSGs the uterus had a T-shaped appearance with widening of its lower two-thirds; in addition, the contrast material showed an irregular, shaggy appearance of the walls of the cavity. In six women the uterus appeared hypoplastic, the length of the cavity measuring less than 2.5cm. Women who had gross cervical changes consistent with in utero DES exposure (described previously) showed significantly more abnormalities on HSGs than those with normal appearing cervices. Of 31 women whose mothers used DES during the first 12 weeks of pregnancy, 22 (71%) had abnormal HSGs and 21 of the latter group showed cervical abnormalities. No relationship was noted between the dose of DES or length of time it was used and radiographic changes.

Haney *et al.*[13] employed HSG to study 13 women with in utero DES exposure and compared them to a control group of 22 women undergoing HSG during infertility investigation. The data revealed a statistically significant finding of a "hypoplastic" appearance of the uterine cavity — the upper uterine segment, endometrial cavity area, and widest diameter of the endocervical canal were significantly smaller in patients in the DES-exposed group.

The clinical significance of these findings and the precise structural changes responsible for them is as yet undetermined, although exposure to DES during Mullerian development is known to cause alterations in the tubouterine configuration.[13]

Singer and Hochman[14] reported an association of an incompetent cervix in a DES exposed offspring. Goldstein[15] noted that of nine DES-exposed patients who conceived and had a congenital deformity and cervical hypoplasia, five developed cervical incompetence during the second trimester. Two of Goldstein's patients were nulliparous, two others had early spontaneous abortions, and one had two spontaneous mid-trimester abortions. Four of the patients were managed successfully during pregnancy with a modified Shirodkar type cerclage. In at least one patient in this group, a preconceptual hysterosalpingogram showed a characteristically widened lower uterine segment. The cause of the cervical incompetence in these patients is unknown. Siegler *et al.*[16] suggest that in these women the fibromuscular junction might be misplaced from the internal os to a lower site on the cervical canal and therefore interferes with the sphincteric function of the internal os. This complication may prove to represent a significant cause of pregnancy wastage in females exposed to DES in utero. Other investigators (Haney *et al.*,[13] Barnes[17]) have not to date shown a statistically significant connection with in utero DES exposure and cervical incompetence.

Bibbo *et al.*[18] found a statistically significant increase in menstrual irregularities among 229 DES-exposed women, compared with 136 age-matched control patients. Oligomenorrhea was the most frequent menstrual complaint in both groups. Age at

menarche was the same for exposed and control patients as well as the mean levels of luteinizing and follicle-stimulating hormones. Haney *et al.*[13] noted high rates of menstrual dysfunction (47%) in their DES-exposed group; chief complaints were of primary dysmenorrhea and oligomenorrhea. There was no adequate control on this reported information.

Barnes and colleagues[19] and Widhoms *et al.*[20] concluded that DES had no major effect on age at menarche or menstrual cyclicity. Rosenfeld *et al.*[21] reviewed reproductive problems in 25 DES-exposed women. Nineteen patients underwent HSG; results were abnormal in 15 instances (79%). The findings were consistent with those upper genital tract abnormalities reported previously by Kaufman *et al.*[12] and discussed above. Rosenfeld's group went further to note a tendency in their patients toward oligo-ovulation (40%); this ovulatory dysfunction has been responsive to clomiphene citrate in restoring ovulation. Abnormal postcoital test results were noted in 75 per cent of patients tested. Cervical mucus was noted in these 15 patients to be scanty, cellular, and tenacious at the time of ovulation (no comparative control group was reviewed). A high incidence of tubal pregnancies was documented in this small group (two in eight conceptions), raising the question of whether there could be structural anomalies of the oviduct or abnormalities of tubal motility related to DES exposure. A 50 per cent frequency of pregnancy wastage in those patients conceiving was noted by Rosenfeld to be consistent with the observations of Berger *et al.*,[22] Kaufman *et al.*,[23] and Cousins.[24]

It is becoming increasingly clear that DES exposed women will experience more unfavorable outcomes of pregnancy than unexposed women. There is a consensus of opinion among investigators[25-27] that there is an increase in pregnancy wastage. In one recent study, spontaneous abortions (26% *vs* 16%), ectopic pregnancy (4% *vs* 1%), prematurity (8% *vs* 4%), and stillbirths (4% *vs* 1%) were all more frequent adverse outcomes in exposed compared to non-exposed DES daughters, respectively. It appears that most DES women do not have trouble conceiving although the uterine abnormalities reported may increase the likelihood of an unfavorable outcome. There is no known adverse effect of pregnancy on adenosis or squamous cell changes in the DES exposed female, and there is no evidence that the potential for malignant change is increased by pregnancy. No information gathered to date has any reliable predictive value in terms of future childbearing or reproductive problems in females with in utero DES exposure.

Effects on Males

Investigations by Bibbo *et al.*[29] and Henderson[30] of males with in utero DES exposure indicated that there was no increase in genitourinary malignancies in these men as compared with non-exposed males. There did appear to be an excess of abnormalities in the genital and urinary tracts in these men. Bibbo *et al.*[29] reported that four of 42 DES-exposed men had epididymal cysts, two had undersized penises, two had small testes, one had a varicocele, and another had a benign testicular mass. In the control group the only abnormal finding was a varicocele in one patient. Gill *et al.*[31] studied 289 DES-exposed males and compared them with 290 control males. They found a combined 30 per cent incidence of abnormal genital lesions — epididymal cysts, hypoplastic testes, and cryptorchidism — as compared to an 8 per cent incidence in the controls. No malignancies have been found in these men to date. No significant differences were demonstrable in the averages of circulating blood hormones assayed.

The findings in males reviewed above are strikingly similar to those described in animal studies. In 1940, Greene *et al.*[32] reported that in male rats the effects of in utero exposure to estradiol or diethylstilbestrol were cryptorchidism, hypoplastic testes, and underdeveloped epididymides. Dunn and Green[33] reported the occurrence of cysts of the epididymis in male mice exposed prenatally to DES. Of the male mice studied, 60 per cent were sterile; 33 per cent had epididymal cysts; 25 per cent had undescended testes; and 25 per cent had nodular enlargement of the seminal vesicles associated with squamous metaplasia.

Gill and his group[31] have performed semen analyses of males with DES in utero exposure. These investigators noted a significant over-representation of low sperm concentrations, low total counts, decreased motility grade, and an increase in the percentage of morphologically abnormal forms as compared with controls. The combination of pathological semen with anatomical abnormalities on physical examination occurred in 60 per cent of the DES-exposed males who had severely pathological semen. It is important to note that these published results on semen analyses by Gill were based on one sperm count and it is well known that two or three sperm counts would be necessary before one could fully evaluate sperm production.

Despite the increase in genitourinary abnormalities in young men exposed to DES in utero, there has been no documentation of any influence on sexual performance.[29] The actual infertility rate in these

men cannot yet be determined nor can the eventuality of developing genitourinary malignancies be ruled out in this group of relatively young men (early to middle twenties).

Screening

In view of the benign (possibly precancerous) and malignant genital abnormalities documented in young females and abnormal genitourinary findings in young males who were exposed to maternal-ingested DES in utero, the need for effective screening is apparent. An elaborate scheme of management of females has been outlined by the Department of Health and Human Services (HHS);[35] no such plan of followup has been suggested yet for males. It has been recommended by the Registry of Clear Cell Adenocarcinoma of the Genital Tract in Young Females that unless a prepubertal child has vaginal bleeding or discharge, pelvic examination can be deferred to the time of menarche or by the age of 14 years. At that time all females with a history of DES exposure should be examined; this should include careful vulvar inspection and vaginal palpation. Inspection of the vagina and cervix is essential prior to cytologic sampling. Cytology is a useful screening measure if anterior, posterior, and lateral walls of the vagina are scraped; this procedure is fraught with a high frequency of false negative findings. Direct visualization of the vaginal and cervical epithelium with iodine staining is mandatory; non-stained areas of the vagina may indicate adenosis. Careful bimanual palpation is always required.

Colposcopy has recently been accepted as being of value in screening DES exposed females for abnormalities. Stafl[36] presents a detailed description of the use of colposcopy in identifying adenosis and possibly early clear cell adenocarcinoma. Stafl notes that the striking difference in the colposcopic findings of DES-unexposed and DES-exposed women is in the extension of the transformation zone between cervical columnar epithelium and squamous epithelium. In DES unexposed women, the transformation zone usually involves only a small portion of the cervix; in DES-exposed women, it was found that the transformation zone is much more extensive, involving both cervix and vagina in 82 per cent of cases examined by Stafl. Colposcopically, Stafl could accurately differentiate between the original squamous epithelium and the well differentiated metaplastic squamous epithelium because of differences in the terminal vesicular network as seen through the colposcope. By visual examination, without the colposcope, Stafl notes that islands of vaginal adenosis seem to be within the original vaginal epithelium; but with the colposcope, adenosis is always present within the transformation zone.

Colposcopy is currently limited because it must be performed by a well trained physician and is a costly procedure. The Department of Health and Human Services recommends that DES exposed females be referred for colposcopy in all cases of abnormal cytology and in cases of extensive epithelial changes.[35] Biopsy of any suspicious areas is then mandatory.

As it is commonly accepted by obstetricians and gynecologists today that DES exposed females who become pregnant do have an increased risk of pregnancy loss and premature birth, it is important that such women be followed carefully during pregnancy. Observation for evidence of premature ripening or dilatation of the cervix is advisable. Cervical cerclage should then be performed when indicated. Hysterosalpingography is not recommended as a routine screening procedure in DES daughters. HHS recommends that it be reserved for cases in which repeated pregnancy loss or infertility occurs.[35]

A sophisticated screening protocol for males exposed in utero to DES has not yet been formulated. The data on the increased incidence of genitourinary abnormalities in these men are of recent determination, and more complete longitudinal studies must be performed. It seems advisable that at present any man with known in utero DES exposure obtain a thorough genitourinary physical examination and appropriate laboratory evaluation. It is important that he and his physician be aware of potential infertility problems due to pathological or decreased sperm. Furthermore only by close prospective followup of these cohorts can there be a determination of any future propensity to develop a malignancy directly associated with prior in utero DES exposure.

References may be obtained from the author.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Diagnosis and Management of Outpatient Adult Asthma

SUSAN K. PINGLETON, M.D.,* *Kansas City, Kansas*

ASTHMA is an exceedingly common disease, in both the pediatric and adult populations. Adult asthma can result from a wider variety of etiologic mechanisms, at times present with less usual symptoms, and unfortunately in some circumstances may be more difficult to treat successfully.

Pathogenesis

Asthma is a syndrome rather than a single disease in which airway smooth muscle contraction or bronchoconstriction is the final common pathway.[1] The basic defect in all asthmatics is simply bronchial hyperreactivity. Normally the caliber of the tracheobronchial tree is a balance between forces tending to cause bronchoconstriction and those tending to cause bronchodilation *(Figure 1)*. When challenged by stimuli such as allergens, irritants, infection or exercise, the tone in normal patients is not upset. Asthmatic patients, on the other hand, have increased bronchial reactivity and when challenged by these same stimuli have symptoms of bronchospasm, cough, and dyspnea.

Many pathophysiologic mechanisms can result in bronchial hyperreactivity in the adult asthmatic. The most well-defined — although the least common single mechanism in adult asthma — is allergic, otherwise known as extrinsic asthma. In this instance asthma is thought to be due to a specific allergen (*e.g.* ragweed pollen). This form of asthma is very infrequent in the adult (probably 10 per cent or less).

The most common etiology of adult asthma is the so-called intrinsic asthma, in which allergy is not involved. Several clinical forms of intrinsic asthma exist.[2] In one form viral respiratory tract infection and bronchitis coexist and appear to cause or exacerbate bronchial hyperreactivity. In these patients, stimulation of bronchial vagal irritant receptors may play a role in the development of asthma. Another clinical form of intrinsic asthma is aspirin-sensitivity asthma or triad asthma. Patients exhibit a triad of nasal polyposis, aspirin sensitivity, and asthma. Development of their bronchospasm is thought to be the result of aspirin inhibition of prostaglandins. Lastly some patients develop bronchospasm after exertion

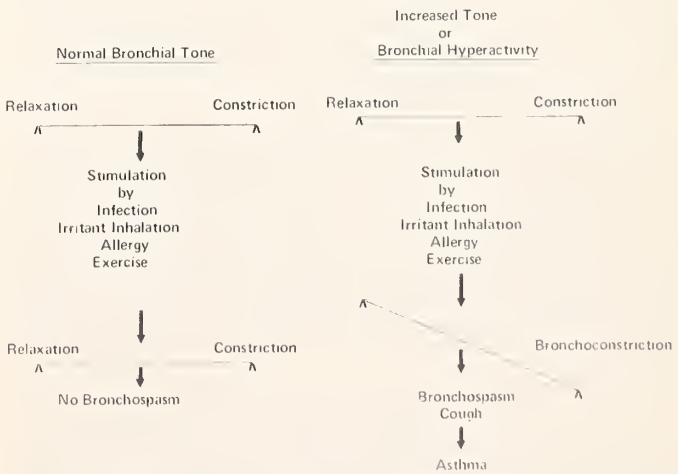


Figure 1. Caliber of the tracheobronchial tree is a balance between forces causing bronchodilation and forces causing bronchoconstriction.

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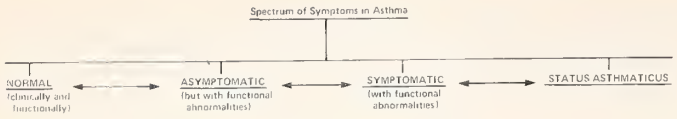


Figure 2. Spectrum of clinical symptomatology in adult asthma.

or exercise. The so-called exercise induced asthma is thought to be due to changes in the respiratory heat exchange system.[3] Hyperventilation during exercise promotes respiratory heat loss and causes bronchoconstriction.

Regardless of the specific etiologic mechanism or clinical form of asthma that exists, we have come to recognize that the clinical symptomatology of adult asthma encompasses a spectrum *(Figure 2)*. Patients may occupy one point of the spectrum or move up and down the spectrum.

Multiple factors can precipitate an asthma attack in the adult *(Table I)*. The history commonly includes a "cold" or upper respiratory tract infection exacerbating asthma. Exposure to an allergen would be an expected precipitating event in patients with allergic asthma. Inhalation of irritants such as smoke, toxic fumes or cold air, as well as exercise and the respiratory maneuvers of laughing and hyperventilation, have been implicated as etiologic factors. In some patients drugs such as aspirin or propanolol are responsible.[4] Finally physicians are beginning to recognize that asthma may be the result of occupational exposure.[5]

TABLE I
PRECIPITATING FACTORS IN ASTHMA

1.	Infection upper respiratory tract pneumonia
2.	Exposure to allergen
3.	Irritant Inhalation
4.	Cold Air, exercise, respiratory maneuvers (laughing, hyperventilation)
5.	Drugs aspirin, propranolol
6.	Occupation

Diagnosis

The classic symptoms of asthma are wheezing and dyspnea. Cough may also be present; indeed, at times it is important to recognize that a dry nonproductive cough may be the sole manifestation of asthma.[6] Basic laboratory evaluation should include chest x-ray, arterial blood gas values, and some form of pulmonary function testing.

Certainly not all that wheezes is asthma. While a good history is of paramount importance in establishing a diagnosis, chest x-ray can aid in narrowing the differential diagnosis, at times provide a precipitating etiology, and demonstrate complications of an acute attack. Followup outpatient chest x-rays probably are not necessary except when the asthma attack is severe.

The basic physiologic abnormality in asthma is expiratory airflow obstruction. A complete battery of pulmonary function studies aids not only in the diagnosis of asthma, but also delineates the severity of airflow obstruction. Not all pulmonary function testing need be done in the laboratory, however. Evaluation of expiratory airflow by measurement of peak expiratory flow rates (Wright Peak Flow Meter) or other simple spirometry can and should be done in the physician's office or emergency room. Slight deterioration from a patient's normal reading can be extremely helpful in diagnosing and treating an early attack. The effect of drug therapy can also be followed easily and yet physiologically.

Arterial blood gas values (ABGs) are the cornerstone of an objective evaluation of a patient's oxygenation status as clinical signs of hypoxemia and hypercarbia are unreliable. Franklin has proposed dividing asthma into five stages based on arterial blood gas determination *(Table II)*.[7] The severity and, therefore, the treatment of an attack is determined by which stage the patient is in. In Stage I (ABGs probably would not be measured), the patient has no subjective limitations and the gas levels would be normal. In Stage II — an early asthma attack — the patient still maintains a normal arterial oxygen level, but only at the expense of hyperventilation (respiratory alkalosis). Stage III represents the stage at which most patients seek medical help. Mild arterial hypoxemia ensues, even with hyperventilation. At State IV, the patient's attack is very severe; airways obstruction is such that alveolar ventilation is compromised and the patient begins to "normalize" the pH and PCO_2. This is an ominous sign. If aggressive drug therapy is not given — or at times in spite of therapy — the obstruction may become more severe, and alveolar ventilation will become more compromised resulting in a PCO_2 elevation and a pH drop. Acute respiratory acidosis in the asthmatic has been associated with sudden death from apnea, cardiac arrhythmia, or cardiopul-

TABLE II
STAGES OF ASTHMA

		paO_2	$paCO_2$	pH
I.	little or no obstruction	N	N	N
II.	mild obstruction (respiratory alkalosis w/o hypoxemia)	N	♦	♦
III.	moderate obstruction (respiratory alkalosis c hypoxemia)	♦	♦	♦
IV.	Severe obstruction (normal pH c severe hypoxemia)	♦♦	N	N
V.	Life-threatening (respiratory acidosis)	♦♦♦	♦	♦

monary arrest. Outpatients with adult asthma commonly present in Stage III.

Therapy

Drugs should be administered in asthma for two reasons — to control symptoms and to decrease the frequency and severity of asthma attacks. As noted earlier, some asthmatics are entirely asymptomatic but demonstrate residual pulmonary function abnormalities (increased lung volumes and airway resistance). Pathologic correlates are mucus plugging, thickened basement membranes, and mucosal edema. The baseline status of lung function plays a primary role in a patient's response to any given stimuli. When an asthmatic is challenged, the severity of an attack can be correlated with the degree of obstruction present before the attack. Patients with residual airways abnormalities, even though asymptomatic, would be expected to have an increased number and more severe attacks. A goal of drug therapy is, therefore, to return the patient not just to an asymptomatic status, but also to a normal functional status, thereby decreasing the severity and frequency of future attacks.

How long should a patient be treated with aggressive drug therapy after an acute attack? McFadden has shown that signs and symptoms of acute asthma resolve very quickly (within hours) after initiation of therapy but physiologic abnormalities may be present much longer (days).[8] Studies in *normal* patients demonstrate pulmonary function abnormalities six weeks after an uncomplicated respiratory tract infection.[9] Asthmatics, with an abnormal bronchial reactivity, would be expected to require even longer to resolve the abnormalities.

Outpatient drug therapy of adult asthma encompasses several classes of drugs. Drugs that enjoy great popularity in the treatment of asthma are methylxanthines or theophylline compounds. A reason for the increase in popularity of these drugs has been the ability to measure the plasma theophylline level. A therapeutic theophylline level is 10-20 μg/ml, the range representing the best balance between efficacy and toxicity. While bronchodilation does exist in levels below 10 μg/ml, maximal bronchodilation with minimal toxicity requires a level of 10-20 μg/ml. Above 20 μg/ml, toxic effects of nausea and vomiting, cardiac arrhythmias, and convulsions have been described.

Oral theophylline compounds are usually initiated in the non-acute stage. An initial dose is 3 mg/kg/6 hr.[10] The dosage is decreased by one-half in patients with liver disease and heart failure. If no adverse reaction occurs but symptoms remain uncontrolled, a total dose of 6 mg/kg/6 hr can be given. Theophylline levels are routinely measured to ensure optimum bronchodilitation and for suspected toxicity. If theophylline levels are not available, the drug should be administered according to the above prescribed dosage. If gastrointestinal symptoms appear, the dosage should be decreased or discontinued. With outpatient theophylline therapy, patient education of adverse side effects is extremely helpful.

A relatively new form of oral theophylline compounds is currently available — sustained release (S-R) tablets or capsules — permitting an eight or 12 hour interval dosage. Sustained-release theophylline compounds such as Theodur, Theolair, and Sustaire are among those preferred by many pulmonary physicians. In some patients the older but still reliable aminophylline tablets administered every six hours are still very effective.

Combination products with theophylline, sympathomimetics, sedatives, and sometimes an expectorant are strongly discouraged as they have a number of disadvantages. Fixed dose ratios prevent proper utilization of each component (especially theophylline). The value of the sedative and expectorant remains unproven.

Sympathomimetic drugs are available via the oral, inhaled, and subcutaneous route.[11] In oral form, ephedrine, terbutaline, metaproterenol, and albuterol are available. While ephedrine has been used widely in the past, its efficacy as a bronchodilator is not as pronounced as newer compounds. Terbutaline, metaproterenol, and albuterol are newer, more effective sympathomimetic bronchodilators. Limiting their use are side effects of tremor, tachycardia and nervousness, which occur in about 10 per cent of patients. While incapacitating in some patients, the side effects may be tolerated in others. Therefore, a patient begining this class of drugs should be informed of the side effects so that, if at all possible, the drug could be continued. Tremors and

tachycardia may diminish after one to two weeks of therapy.

Sympathomimetics may be inhaled either from a hand-held nebulizer or a metered dose device. Controversy exists concerning the safety of metered dose sympathomimetics relative to the potential for abuse in the setting of acute asthma. The advantages of metered dose drugs are portability and therapeutic effectiveness. Metered dose sympathomimetics have a place in chronic drug therapy. With only a few exceptions (*e.g.* exercise-induced asthma), they should not, however, be the sole therapeutic agent. Patients should be educated regarding the danger of inhaler abuse. They must recognize that an increased use represents deterioration of lung function, and more aggressive therapy prescribed by a physician is needed.

Subcutaneous sympathomimetics (epinephrine, terbutaline) are reserved for the acute attack. Care must be taken in administering these drugs subcutaneously to patients with a history of coronary artery disease.

Corticosteroids are extremely effective bronchodilators.[12] In the outpatient setting, oral steroids (usually prednisone) are most often given after failure to control symptoms by theophylline and sympathomimetics or when deterioration occurs in patients on maintenance theophylline and sympathomimetics. The most efficacious type and dosage of oral corticosteroids varies with the physician's personal experience. Prednisone, 40-60 mg/day, is the form and dosage utilized by this author. Rapid taper (1-2 weeks) is performed with symptomatic and functional improvement.

Recently an inhaled corticosteroid, beclomethasone dipropionate, has become available with the advantage of maximum bronchodilation and minimum steroid side effects. Rational use is in steroid-dependent asthma where beclomethasone may allow significant decrease or even discontinuation of oral steroids. When attempting to decrease or discontinue oral steroids, beclomethasone must be overlapped or taken concomitantly with the steroids for at least one to two weeks. The dose is two puffs (100 mg) four times daily. Side effects include oropharyngeal candidiasis, which may be minimized by gargling with water or mouthwash after each use.

Two other drugs deserve mention although one (cromolyn) may play only a limited role and the second (atropine and atropine-like compounds) have not been released for general use at this time. By inhibiting chemical mediated release from sensitized mast cells, cromolyn should have efficacy in those patients with allergy as a mechanism of asthma. Cromolyn is a prophylactic drug; its use is contraindicated in an acute attack. Regular inhalation (every 6 hours) must occur for the drug to be effective. Cromolyn should be considered only in those patients whose symptoms can be historically related to a specific allergen. As a whole, cromolyn has been disappointing in adult asthma. Anticholinergic drugs (atropine or atropine compounds) may prove useful in those asthmatics whose bronchospasm is precipitated by cholinergic mechanism.

The role of antibiotics in the treatment of asthmatic attacks precipipitated by upper respiratory tract infection is controversial. Even in the absence of a specific infectious process (*i.e.* pneumonia), broad spectrum antibiotics (ampicillin, tetracycline, erythromycin) may decrease the length of an attack.

Drug therapy for any given patient with asthma is determined by the severity of the patient's disease and his responsiveness to therapy. Specific recommendations are difficult but generalizations can be made *(Table III)*. Mild asthma can most often be controlled by a single agent taken regularly. Therapy is most often begun with a theophylline compound. If attacks continue, an oral sympathomimetic, which acts synergistically with the theophylline compound, is added. Aerosol sympathomimetics are added if further symptom relief is needed or with symptom deterioration in an acute attack. In the appropriately selected patient, a one month trial of cromolyn may be instituted. Moderately severe asthma requires a theophylline compound, oral sympathomimetics, and oral steroids (prednisone). Rapid steroid tapering is the goal; however, this must be balanced against the patient's symptoms. The addition of beclomethasone may allow steroid tapering. An acute attack of severe asthma unre-

TABLE III
DRUG THERAPY OF ASTHMA BASED ON SEVERITY

Mild asthma	
	:regular administration of theophylline
	:if symptoms uncontrolled add oral sympathomimetic
Moderately severe asthma	
	:theophylline, sympathomimetic, oral corticosteroids
	:Beclomethosone to substitute when oral steroids tapered
	:trial Cromolyn when indicated
Severe asthma	
	:intravenous theophylline, inhaled sympathomimetic (via hand-held nebulizer), intravenous corticosteroids

sponsive to subcutaneous epinephrine has been classified as status asthmaticus. In this instance hospitalization is required with treatment with intravenous bronchodilators.

One other specific clinical situation deserves mention. Occasionally patients are seen with dyspnea and a non-productive cough. In spite of the absence of bronchospasm, the diagnosis of asthma needs to be strongly considered.[13] Pulmonary function testing may be completely normal unless a cholinergic challenge is included. Methacholine, a drug that stimulates cholinergic receptors, will cause a 10-20 per cent decrease in the forced expiratory volume in one second if the patient has hyperreactive airways. The therapeutic implication of such a diagnosis is evident. Cough suppressors or expectorants will not be helpful. Instead the patient should be treated with bronchodilators, usually a sympathomimetic. As anticholinergic drugs become available, this may be the appropriate situation for their use as bronchial irritant receptor stimulation of cholinergic receptors is suspected as the pathophysiologic mechanism.

In summary, diagnosis of the adult asthmatic is made with symptoms of dyspnea, bronchospasm, or chronic cough. Treatment requires bronchodilator drugs to relieve symptoms and decrease the number and severity of acute attacks.

Self-Assessment Questions

1. Etiologies of asthma include:
 a. allergy
 b. exercise
 c. drug sensitivity
 d. upper respiratory tract infection
 e. all of the above
2. A 25-year-old, otherwise healthy, white male presents to the physician's office with chronic, non-productive cough that worsens with exercise (jogging). History, physical examination, and routine laboratory tests (including chest x-ray and spirometry) yield completely normal results. The next diagnostic step would be:
 a. barium swallow
 b. fiberoptic bronchoscopy
 c. repeat spirometry with methacholine challenge
 d. no further diagnosis needed
3. Appropriate treatment of the patient in *Question 2* would include:
 a. expectorants
 b. bronchodilators
 c. antibiotics
 d. no treatment; followup in one month
4. A 30-year-old white female known to be asthmatic appears in the physician's office with complaints of wheezing and dyspnea. Physical examination reveals her to be extremely dyspneic and tachypneic with use of accessory muscles; marked wheezing is noted throughout all lung fields. Room air arterial blood gases show PaO_2, 60 mmHg; $PaCO_2$, 45 mmHg; and pH, 7.4. These findings would be interpreted to show:
 a. stage I asthma
 b. stage II asthma
 c. stage III asthma
 d. stage IV asthma
 e. stage V asthma
5. Appropriate therapy for the patient in *Question 4* would include:
 a. intensive bronchodilator therapy
 b. antibiotics alone
 c. expectorants

(Answers on page 649)

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Drug INFORMATION

LINDA HOGAN, M.S., R.Ph., *Editor*

Transdermal Nitroglycerin Patches

Question: What is the newest administration method for topical nitroglycerin?

Answer: The U.S. Food and Drug Administration has approved three nitroglycerin-containing adhesive patches for use in the treatment of angina pectoris. They are: Transderm-Nitro (Ciba), Nitro-Dur (Key Pharmaceuticals), and Nitrodisc (Searle). These patches produce plasma concentrations comparable to those obtained with nitroglycerin ointment.[1]

There are several advantages offered by the patch system over nitroglycerin ointment. First, they are easier to apply. The patch is placed on a hairless area of the upper arm or chest. No measuring or manipulation is required. Second, they have a longer duration of activity. The patches deliver the drug continuously for 24 hours; ointment offers anginal relief for four to eight hours.[2] Third, because the patch does not require patient manipulation of dose, the amount of nitroglycerin administered is more consistent. With ointment, plasma levels vary with both the amount applied and the surface area of application.[3]

There are several differences among the various patch systems. Most notable is the mechanism of drug delivery; however, its relationship to therapeutic efficacy has not been determined. Transderm-Nitro has a semipermeable membrane which controls the amount as well as the rate of drug release. It is the only system that directly controls the rate of release, which may be important in highly permeable skin.[4] The other systems utilize drug-polymer matrices that allow only a limited amount of drug to be available for delivery at any one time. All of these systems rely on passive diffusion along a concentration gradient to drive the drug from the patch through the stratum corneum into the blood supply.

In addition, each system has different labeling which can cause confusion. Transderm-Nitro is labeled in terms of amount of drug delivered: Transderm-Nitro 5 releases 5 mg/24 hrs. Nitro-Dur labeling is based on surface area: Nitro-Dur 10 cm has a surface area of 10 cm, but releases 5 mg of drug/24 hrs. Nitrodisc is labeled in terms of total nitroglycerin content of the patch: Nitrodisc 16 mg contains a total of 16 mg of drug, but releases approximately 5 mg/24 hrs. The most pertinent information for the clinician is the amount of nitroglycerin released in vivo/24 hrs.[5] These labeling patterns are confusing to clinicians when determining dosage for patients. Proposed changes not only would change the statement of drug dosage, but would identify the individual patches as well.

To date, there have been no published clinical trials comparing these systems. Until proven otherwise, these systems cannot be assumed to be directly interchangeable.[6] With any of these systems, the dosage must be individualized.

Submitted by
Jeanne Mullen, R.Ph.

References are available from the Drug Information Service, Dept. of Pharmacy, 3930 Cambridge, Kansas City KS 66103.

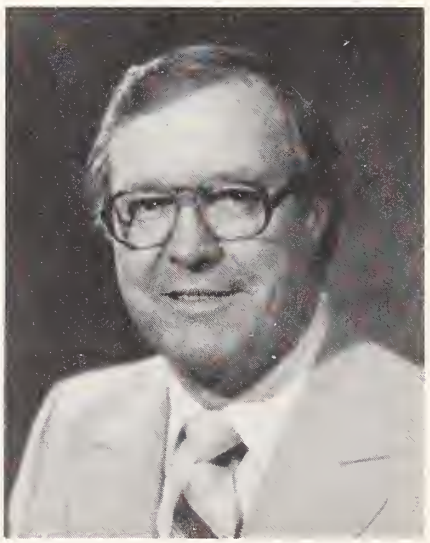
The President's Message

Dear Colleague:

The elections are over. Some candidates tasted victory, others the sting of defeat. Hopefully many of our members were involved, to varying degrees, in these campaigns.

This is the time to contact your legislator and offer your expertise and willingness to help with legislation that impacts on health. Unfortunately there are very few in the legislature who have any affiliation with medicine or physicians. Thus many decisions to determine who will practice medicine and how it will be practiced will be made by non-medical people — farmers, lawyers, teachers, and business people. In order to have an influential impact, we as physicians must make our concerns known and be prepared to show valid reasons for our views.

The legislative session is just a few weeks away. Our legislative committee, under the experienced leadership of Dr. Jim Gleason, is preparing to represent your views on health issues. We urge all members to follow our legislative bulletin so that you will be informed as to the issues and their status as they pass through the legislative process.



Let your committee and your legislator know your views. You can and should be involved in decisions that impact on your destiny.

President



Academic Question

The process of medical education, never notably tranquil (at least since the days of Abraham Flexner), has been experiencing intermittent clonus and tonus of late, prompting the usual introspective therapies directed toward producing new methods. This is not surprising, given the changes in medical substance and purpose (not to mention the social climate) over the years but calls to mind that the whole subject of education has never been easily contained. It is one of those elusive concepts — known to all and easily understood until one attempts to confine it within explicit and reproducible terms of constant meaning to all who speak of it. Like beauty, it comes to lie in the eye of the beholder.

A point in this regard is made by Somerset Maugham in his story about the verger of St. Joseph's Church whose years of faithful and exemplary service are brought to an abrupt halt when the new vicar learns that the verger can neither read nor write and — well, we really can't have that now, can we? The verger's simple needs and abstemious habits (save his one indulgence, his cigarets) have, however, permitted him to put aside a little money and he is able to negotiate the proprietorship of the small tobacco shop he has patronized. The same qualities result in the prospering of the shop — in fact, over a period of years, the acquisition of several shops.

This results in the development of a bank account sufficient to attract his banker's attention. The banker approaches him regarding the possibility of putting the money to more active use than a simple account. In the course of the discussion, the banker is astounded to learn of the man's illiteracy and reacts with the not unexpected question: if he has done so well in spite of this, what might he have been had he not have been so disadvantaged? The man responds, not without a smile, that he presumes he would still be the verger of St. Joseph's.

Literary contrivance acknowledged, Maugham exposes the question of the character of education. Educational tenets range from advocacy of storage of information by rote to the conviction that it amounts primarily to knowing where to find information when the need arises. The former has the merit that the acquisition of some information by forced feeding is inevitable and this is a simple matter of extension. The latter has merit in the implication that some education is necessary to define the information needed as a prerequisite to the effort to find it. It can include those who do not have the knowledge but have the manipulative ability to use those who do have it. Between these extremes, each of which can claim some form of success if one fits it to an appropriate structure of conditions (which is the usual technique of theories), lies the world of education.

A — perhaps *the* — problem with education is that, like all intellectual exercises, it is not a concise and palpable body. It exists within the complexity of experiences and mental explorations that one age or group tries to pass on to the next, but it is not a free standing entity. What we channel into each other is integrated with all else within us, sometimes to mutual benefit, sometimes not. Consequently, new theories and different methods appear at intervals, exciting predictably mixed responses. The accumulation of "new" information continues in spite of such distractions. Some is recorded and not recognized for its value at the moment or other conditions have not reached the point where it can be used effectively. Never lacking in ambition, education seeks to encompass the whole process for purposes of communication which is, of course, education in the active voice.

No discipline (another handy term to try to delimit an abstract concept while endowing it with virtuous connotations) is required to reconcile tradition and experience with a coalition of developing and changing ideas more than medicine. As it tries to be all things to all people, medical education seems to give the impression of uncertainty or even lack of rele-

vance to students because it is in this constant state of reexamination. Fortunately, the mass of medical knowledge constitutes a force that presses into the candidates enough of the essential materials that they can begin to function in practice, and the individual then places his or her own stamp of character on its future form. The educational state of the profession comes then to be judged by the total effect. But methods must be continually shifted, altered, stretched, and compressed to "improve" or at least meet current requirements. The real changes are, in fact, cause and effect at the same time. Medicine has undergone a revolution in the past century (by no means finished yet nor will it seemingly ever be). Students of today learn by different methods than did their grandparents — there seems to be much more to learn since additions are made faster than the obsolete can be removed (or even recognized as obsolete) but they end up talking very nearly the same language — since the subject is the same.

The justification for pursuing the matter in the present context is the inescapable fact that medical information has, for some time, been accumulating at an overwhelming rate (to which we hope we have contributed). This is not to say that education has been increasing at the same rate since it tends to lag behind but the process does incite various efforts toward making the transformation of information into education more efficient. Attempts to weed out the unnecessary have led to the question, what is the irreducible minimum of medical basics which must be retained in the face of burgeoning detail? And when is the best time to present those basics?

The traditional concept has been, of course, to take a properly prepared student, apply medical basic sciences in concentrated doses, combine with clinical experience of more or less length, temper with hospital experience, and offer to an eager but critical public. The expectation has been that the individual would emerge with a determination to continue intellectual growth, an expectation which has been called into question of late, leading to pressurized processes to demonstrate that this is fact. This call for continuing education has been due less, we suggest, to any significant lack of proficiency (that can be remedied in this manner, at least) than it has been the lack of awareness of the public as to what all this curricular maneuvering had produced and the extent to which physicians voluntarily continued to pursue them. CME is largely a defensive expression designed to impress the unknowing that physicians do continue their efforts to keep educated. Consequently, it has been demonstrated that such programs have not changed physicians' habits as much as they have provided consumers with more publicized evidence of the activity. As far as learning is concerned, it is entirely possible, if one is so inclined, to meet the requirements for approval with complete exclusion of education, although the process may be continuing and medical. Thus, there is reasonable question as to whether the real intent of such programs is being realized more proficiently than before the travel agents took over.

This growth of medical information has had a part in changing the form of medical practice as well. No small factor in the growth of the era of specialization has been the realization that, however desirable the basic indoctrination into medical practice, no one could claim specialized expertise in all directions. The restriction of some practitioners to specialized fields was a logical step, accelerating advances in them and providing expert (sometimes too narrowly so) teachers of them. With the inevitable pendulum effect, there has been a shifting away from this fascination with the purified specialty as a more generalized approach has gained new respect, supported as it has been by a combination of increased general training (not just by specialists but by those actually practicing it as well) and judicious improvement of image. More recently, this intrinsic accommodation to the growth of information has been expressed no less by the extrinsic development of various categories of assistants — extensions of the physician in the accomplishment of the increasing variety and sophistication of services. While the physician is not relieved from the responsibility of continued education, the application and execution of that education can be shared and expanded with (it is hoped) overall physician control.

At the same time, the electronic age has introduced a beguiling assistant in the form of the computer which in a sense might be considered the embodiment of that principle of education noted above: the ideal repository for all that information one can't hope to retain personally but can introduce into this handy receptacle of probably unlimited capacity. It would seem this is perilously close to confirming that philosophy that education resides in the knowledge not of the facts but where to find them. It suggests that the graduate of tomorrow may be sent out with a computer and the appropriate supply of software (to which additions will be made by contract), ready to perform his ministrations by keyboard based on a print-out of information passed in from the front office where a prepared questionnaire has recorded it. Given the average physician's love of gadgetry, the concept has a certain potency

(Continued on page 649)



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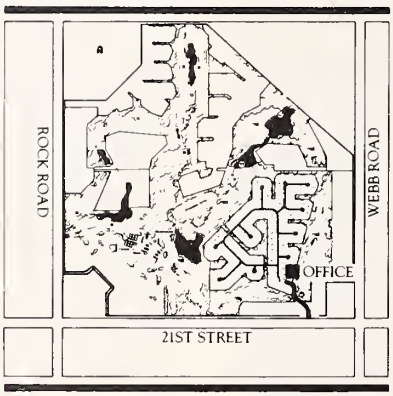
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Outpatient Adult Asthma

(Continued from page 643)

Answers

1. e
2. c
3. b
4. d
5. a

Academic Questions

(Continued from page 647)

but the profession will undoubtedly be saved when the physician finds it is no more satisfying to abdicate his responsibility to the box and screen than to other human agents.

Still, the computer industry, as it has introduced us to bits and bytes and excited our fancies of a computerized world allowing its inhabitants the bliss of total indolence, has promoted an admonition that comes close to being the most durable educational philosophy. We are reminded that human programming is subject to the same hazard as computer programming: garbage in, garbage out. — *D.E.G.*

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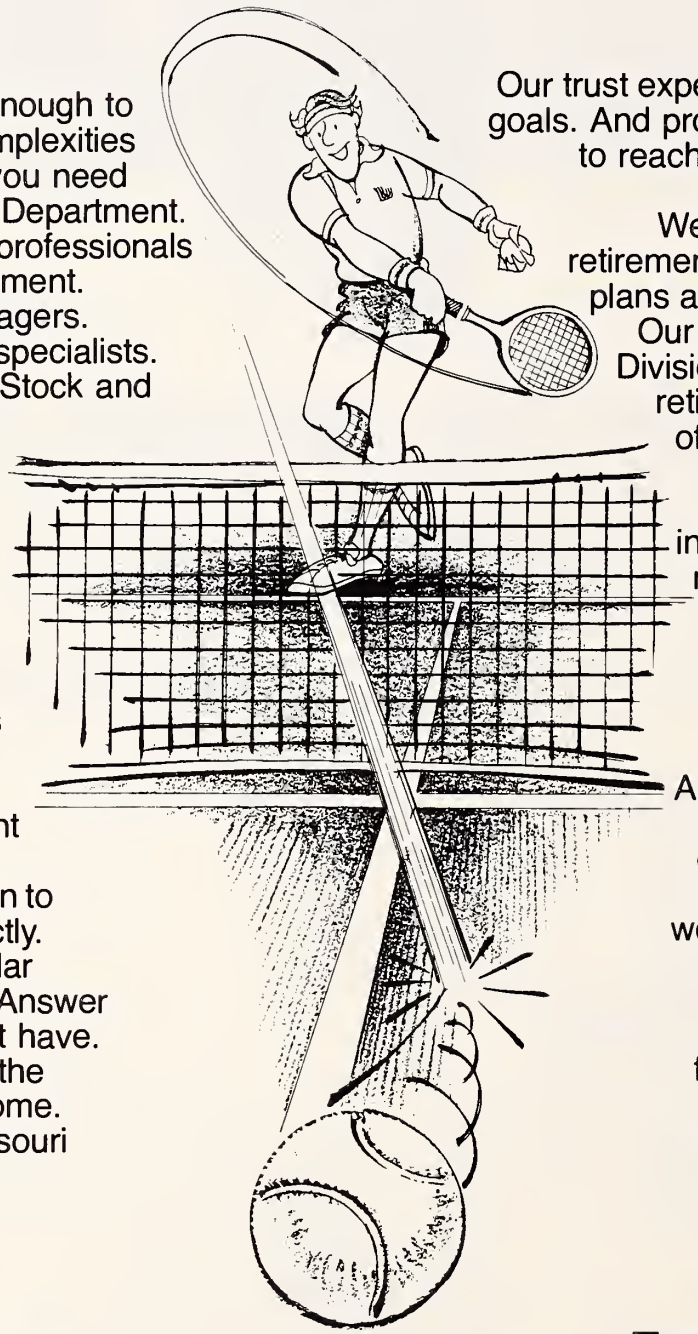
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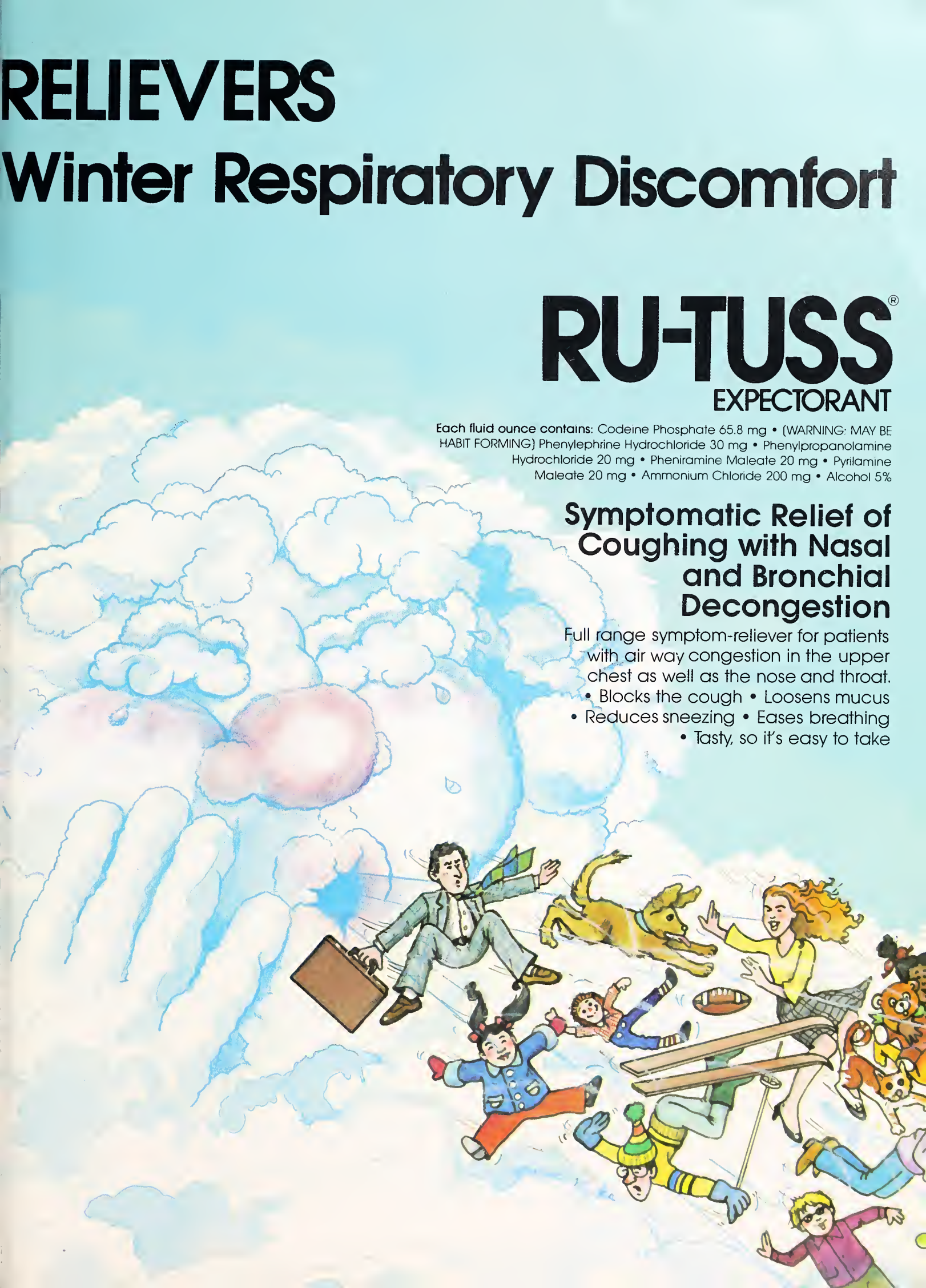
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Ru-Tuss Expectarant is an aral antitussive, antihistaminic, nasal decongestant and expectorant preparatian.

INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated far symptomatic relief af upper respiratory congestian associated with pharyngitis, tracheitis, bronchitis, and allergic rhinitis. Also, for the temporary relief of symptoms assaciated with hay fever, allergies, nasal congestian and caugh due ta the cammon cold.

CONTRAINDICATIONS Hypersensitivity to antihistamines. Cancamitant use of an antihypertensive or antidepressant drug containing a monaamine oxidase inhibitar is contraindicated.

Ru-Tuss Expectarant is contraindicated in patients with glaucama, bronchial asthma and in wamen who are pregnant.

WARNINGS Ru-Tuss Expectarant contains codeine phasphate, therefare, the patient should be warned of the patential that this drug may be habit forming. Ru-Tuss Expectorant may cause drawsiness. Patients should be warned of the passible additive effect caused by taking antihistamines with alcahal, hypnatics, sedatives and tranquilizers.

PRECAUTIONS Patients taking Ru-Tuss Expectarant should avaid driving a motor vehicle ar operating dangerous machinery (See Warnings). Caution should be taken with patients having hypertension, diabetes, hyperthyroidism and cardiovascular disease.

Caution should also be used in patients with pulmonary, hepatic or renal insufficiency.

ADVERSE REACTIONS Ru-Tuss Expectarant may cause drowsiness, lassitude, giddiness, dryness of mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervausness, and insomnia. Overdoses may cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stupor, tachycardia and even convulsions.

DOSAGE AND ADMINISTRATION Adults: 1 or 2 teaspoonfuls, orally, every 4 hours, not to exceed 10 teaspoonfuls in any 24-hour period.

Children 6 to 12 years of age: ½ the adult dose, not to exceed 6 teaspoonfuls in any 24-hour period. Children 2 to 6 years of age: ½ teaspoonful every 4 hours, not no exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age: Use as directed by a physician.

HOW SUPPLIED: (16 fl. oz.)

Pint Bottles	NDC 0524-1010-16

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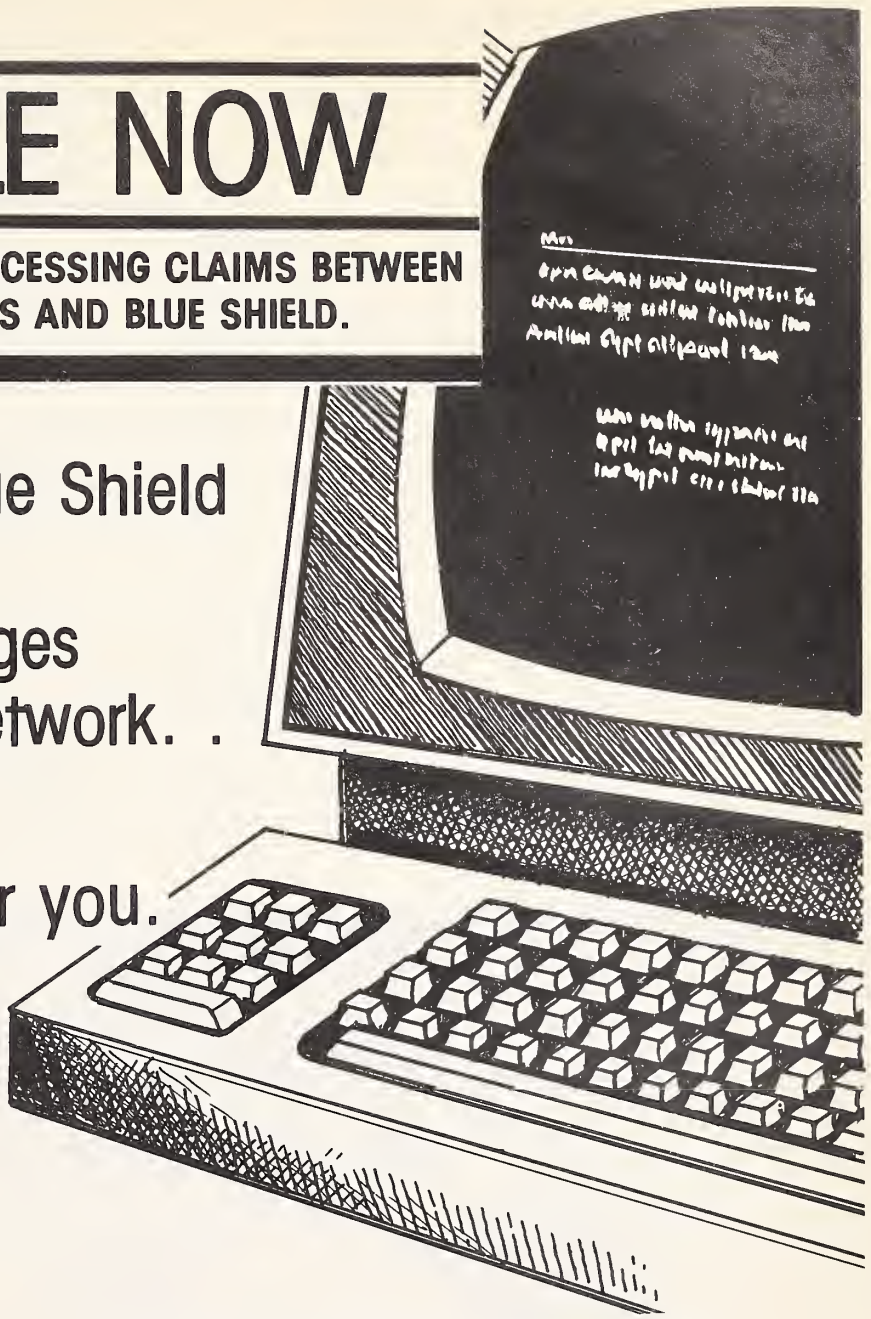
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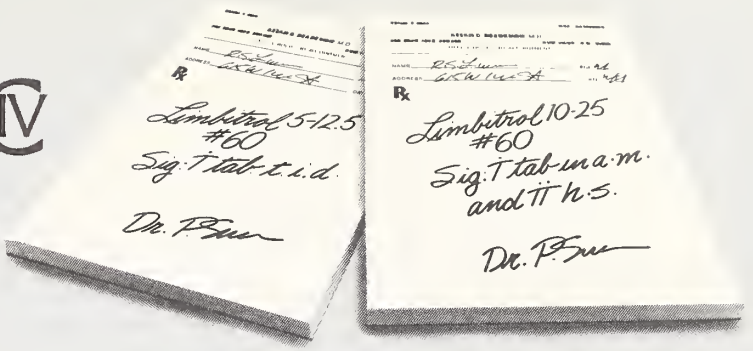
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Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)
Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)



In anxious depression,

SPECIFIC FOR THE NONPSYCHOTIC PATIENT

Fits the picture of anxiety/depression correlation

Most patients with a mood disorder have a mixture of anxiety and depression. One clinician[1] found a correlation of 0.7 in anxiety and depression scores; another[2] has estimated that 7 of 10 nonpsychotic depressed patients are also anxious. For the dual symptomatology of anxious depression, Limbitrol provides dual medication.

More appropriate for the nonpsychotic depressed and anxious patient

Limbitrol contains both amitriptyline, specific for symptoms of depression, and a benzodiazepine, specific for the symptoms of anxiety. Thus it is a better choice than other dual agents for anxious depression that contain a phenothiazine, a class of antipsychotic drugs less specific for anxiety and now generally avoided in nonpsychotic patients.[2,3]

Avoids the risk of tardive dyskinesia carried by the phenothiazine combinations

The causal relationship between the phenothiazines and other extrapyramidal side effects, including tardive dyskinesia, is well established. In contrast, the reported incidence of these adverse reactions with Limbitrol or either of its components is rare.

References: 1. Claghorn J: *Psychosomatics 11*:438-441, Sept-Oct 1970. **2.** Rickels K: Drug treatment of anxiety, in *Psychopharmacology in the Practice of Medicine,* edited by Jarvik ME. New York, Appleton-Century-Crofts, 1977, p. 316. **3.** Baldessarini RJ, Tarsy D: Tardive dyskinesia, in *Psychopharmacology: A Generation of Progress,* edited by Lipton MA, DiMascio A, Killam KF. New York, Raven Press, 1978, p. 999.

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving).

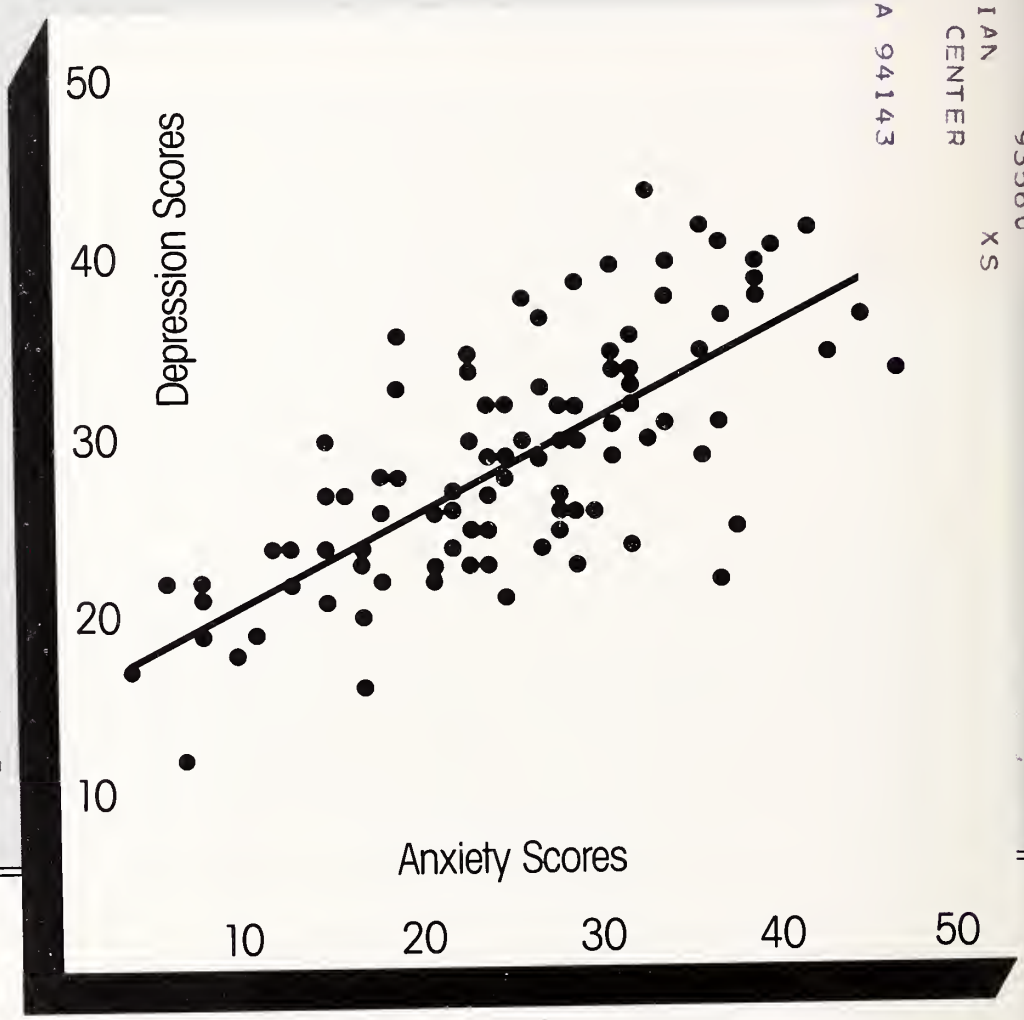
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100, Prescription Paks of 50

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MORE DEPRESSION MEANS MORE ANXIETY...



The graph illustrates the close correlation between depression and anxiety derived through the MMPI and the Taylor Manifest Anxiety Scale in 100 nonpsychotic psychiatric patients. The Coefficient of Correlation is 0.7. As depression increased, so did the anxiety levels.
—Adapted from Claghorn J[1]

A key reason why

MORE PHYSICIANS ARE CHOOSING LIMBITROL®

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)
Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

1. Claghorn J: *Psychosomatics 11*:438-441, Sept-Oct 1970

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